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Monthly Message

In the London *Daily Telegraph and Morning Post* of Wednesday, 9 July 1958, there was a half-column article with prominent headlines, "Boy Left for 20 Minutes After an Operation; Coroner Critic." The article then described a boy of 16 who was left unattended in a side ward following a tonsillectomy: 20 minutes later he was dead. "A verdict of death by misadventure was recorded on the board . . . Dr. Gilbert Forbes, pathologist, said death was due to respiratory failure from an unknown cause." In the course of the article it was stated that the anesthetist said he took no responsibility for the patient after handing him over to the nursing staff. The coroner's comment, "I would have thought the common-sense view was that the person responsible for making a person unconscious should have been responsible for him until unconsciousness had worn off."

At the time he was placed in the ward his condition was described as good, with full and regular pulse, and the anesthetist expected him to be watched, and proper measures taken to ensure a clear airway. The nursing staff stated that there was no particular reason for the boy to have been put in the side ward. The nurse in charge took a look at him and then went to lunch. When she came back he was dead. The assistant head of the nursing office said that although ideal, it was not always practicable to watch all patients recovering from an anesthetic, and stated that could be left to the Sister in charge.

The above act emphasizes the importance of personal responsibility. The more a municipality, state, or Federal government dilutes that sense of responsibility, the more the sense of personal care and responsibility dwindles. These faults are so easy to justify: "It wasn't my job." Every doctor must always accept decision and personal responsibility, and owes it to his patient to treat him as he would like to have himself or a member of his family treated. Whether it be for simple vaccination or major surgery, he should ensure that every safeguard possible has been provided and not leave it to the enfolding arms of the organization.

Frank B. Berry

FRANK B. BERRY, M. D.
Assistant Secretary of Defense
(Health and Medical)

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Foreword

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Assistant Secretary of Defense (Health and Medical) and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal.*

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UNITED STATES ARMED FORCES
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PALPABILITY OF THE LIVER EDGE IN HEALTHY ADULTS

EDDY D. PALMER, *Lieutenant Colonel, MC, USA*

UNCERTAINTY over just how far below the costal margin the right lobe of the adult liver may normally extend seems to be common. Judging from requests for gastroenterologic opinion, discovery that the liver edge descends a centimeter or two below the right costal margin is rather widely considered a sign of liver abnormality. Perhaps one reason is that most textbooks are noncommittal about the normal location of the inferior hepatic edge or ignore it completely. A few encourage distrust of the palpable liver: "Usually the presence of a palpable liver edge below the costal margin indicates either ptosis or enlargement"[1]; ". . . the lower border (of the liver normally) extends to the edge of the right thoracic cage."[2]

The location of the lower edge of the liver does not, of course, necessarily bear a relationship to the size of the organ, and obviously one must determine the position of the liver's cephalad limit by percussion before hepatomegaly can be diagnosed. Nevertheless, whatever the liver's size, its upper surface is remarkably constant in its location, and diffuse hepatomegaly manifests itself almost wholly in the downward direction. Conversely, any great degree of extension of the liver's edge below the costal margin almost always indicates hepatomegaly. The main exceptions are found in cases of severe pulmonary emphysema and in true ptosis of the liver. This last phenomenon can exist only if hepatophrenic contact has been broken and the liver's suspensory ligaments are greatly elongated.

The present study was made to determine the range of positions occupied by the inferior border of the liver's right lobe,

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as judged by palpation, in a group of normal adults. It seems strange that, in this day of isotopic medicine, information is so very sparse on this fundamental point of physical anatomy.

MATERIAL

Observations on the liver were recorded during personally conducted, complete, routine physical examinations of military personnel, and data were compiled on 1,000 subjects who were judged to have no liver disease. Most of the examinations were either annual physicals or separation physicals, with fewer reenlistment, preflight, special assignment, or retirement physicals. Personnel who indicated suspected illness involving the torso were excluded. Similarly, an occasional patient who was not able to relax sufficiently for a satisfactory abdominal examination had to be excluded.

It was, of course, essential that instances of liver disease be recognized and eliminated. A negative history and, when the liver could be palpated, a sharp, smooth, and normally firm edge were insisted on for inclusion in the study. In addition, liver function tests were carried out on subjects with the larger livers, and needle liver biopsy specimens were obtained in 11 cases about which there was some question of normalcy. Some cases of chronic liver disease were recognized in the course of these routine physical examinations[3] and were excluded.

POSITION OF INFERIOR HEPATIC MARGIN

Special attention was paid to examination of the liver, and there is no question but that many of the potentially palpable livers would have been overlooked if this had not been a specially directed study.

The lower edge of the right lobe of the liver which is normal or is diffusely enlarged parallels only in a very rough way the line of the right costal margin. Near the body's midline the liver edge swings across to the left at a level which is quite far below the xipho-gladiolar junction. Normally, then, the edge and surface of the liver are exposed well below the rib cage, within the subcostal angle, and this is so even during forced expiration. Because in this area the liver is overlaid by the rectus muscles and associated tendons, however, palpation here for judgment of liver size and consistency is notably unreliable.

It is necessary, then, to be very precise about the point at which the measurement is made when recording liver size on the basis of the position of its edge. The best point for the determination lies along a line just lateral to the right rectus muscle margin, in the linea semilunaris. This strictly anatomic criterion for the vertical line of palpation is considerably more consistent as a guide, regardless of body build, than is the midclavicular or nipple line. Even in very flabby people the linea semilunaris can usually easily be located by having the supine patient raise

his head while keeping his shoulders where they are, and this is a good first maneuver for palpation of the liver.

The measurement must be made at the peak of the deepest inspiration that the subject is able to take, when he is relaxed, with thighs and knees flexed. It is important that the examiner assure himself that the subject has made a maximum effort and, most important, that the inspiration has been made with the mouth open. Almost all people find that, after a full breath has been taken through the nose, merely opening the mouth permits noticeable further descent of the diaphragm.

As for the mode of palpation itself, it can only be said that scriptive comment is wholly insufficient—every doctor must find out for himself the way of using the hand which for him proves most fruitful. Probably the two commonest mistakes in technic are too heavy palpation and beginning palpation at the costal margin rather than far down on the abdomen. Some young men, it must be noted, seem to lift the chest wall off the liver at the end of inspiration, and this must be recognized so that palpation will be sufficiently heavy and deep. Confusion of a tendinous inscription with the liver edge is a possibility when the subject is muscular, as palpation is carried from the linea semilunaris over the rectus muscles. An especially important habit is to make several palpatory efforts when the liver cannot be felt. It is strange, indeed, how three or four successive efforts may fail to reveal the liver edge, while the next, made in the same location, does so easily.

FINDINGS

When examined by the technic described, the liver edge in 42.6 per cent of the subjects could not be palpated, in 14.4 per cent it could be felt to descend to the costal margin but not beyond; in 15.0 per cent it descended 1 cm below the costal margin; in 12.9 per cent it descended 2 cm; in 8.2 per cent, 3 cm; in 5.7 per cent, 4 cm; and in 1.2 per cent, 5 cm. Separation of the second category—the situation in which the edge can be felt to descend as far as but not beyond the costal margin—may seem a bit artificial because of the rather fine point in examination interpretation it interjects; nevertheless, it is being used because, of course, detection of the liver's lower margin in such cases eliminates immediately the possibility of liver atrophy.

In tables 1 through 5 the position of the liver's lower edge is correlated with the subjects' sex, age, height, weight, and configuration of subcostal angle. The last was designated arbitrarily at the time of each examination as medium (553 cases), narrow (225 cases), and wide (222 cases), on the basis merely of general impressions of and experiences with subcostal angles. It is clear that none of these factors by itself had a controlling influence over liver size, and when the individual data were reviewed no combination of possibly controlling factors was

TABLE 1. *Palpable size of liver correlated with sex in 1,000 subjects*

Sex	Number of subjects	Relation of liver edge to costal margin (per cent of subjects)						
		Not palpable	At margin	1 cm	2 cm	3 cm	4 cm	5 cm
Men	717	39.7	15.9	12.6	15.1	8.4	6.7	1.7
Women	283	49.8	9.9	21.2	7.4	7.4	3.2	1.1

TABLE 2. *Palpable size of liver correlated with age in 1,000 subjects*

Age (years)	Number of subjects	Relation of liver edge to costal margin (per cent of subjects)						
		Not palpable	At margin	1 cm	2 cm	3 cm	4 cm	5 cm
17 - 20	141	38.3	14.2	13.5	8.5	11.3	9.9	4.2
21 - 25	292	34.9	14.8	17.5	15.4	9.2	8.2	0
26 - 30	172	63.8	6.3	7.5	15.5	5.2	0.6	1.1
31 - 35	105	42.9	20.0	8.6	17.1	11.4	0	0
36 - 40	142	42.6	12.8	17.0	10.6	9.9	5.0	2.1
41 - 45	84	46	4	21	14	5	6	4
46 - 50	39	15	49	28	0	7	0	0
51 - 55	10	60	40	0	0	0	0	0
56 - 63	15	27	13	40	7	0	13	0

TABLE 3. *Palpable size of liver correlated with height in 1,000 subjects*

Height (inches)	Number of subjects	Relation of liver edge to costal margin (per cent of subjects)						
		Not palpable	At margin	1 cm	2 cm	3 cm	4 cm	5 cm
57 - 58	8	75	25	0	0	0	0	0
59 - 60	15	27	13	40	20	0	0	0
61 - 62	31	48	13	19	3	7	10	0
63 - 64	87	38	21	0	24	7	10	0
65 - 66	186	41.9	4.8	30.6	8.1	9.7	1.6	3.2
67 - 68	238	35.4	20.3	10.1	15.2	11.4	5.1	2.5
69 - 70	188	52.4	14.3	12.7	11.1	4.8	3.2	1.6
71 - 72	141	42.6	12.8	17.0	4.3	12.8	10.6	0
73 - 74	84	39	14	10	25	0	12	0
75 - 76	18	67	0	6	27	0	0	0
77 - 78	4	0	75	0	25	0	0	0

TABLE 4. *Palpable size of liver correlated with weight in 1,000 subjects*

Weight (lb)	Number of subjects	Relation of liver edge to costal margin (per cent of subjects)						
		Not palpable	At margin	1 cm	2 cm	3 cm	4 cm	5 cm
90 - 110	84	57	14	16	6	7	0	0
111 - 130	131	50.4	14.5	18.4	11.7	1.5	3.5	0
131 - 150	283	40.3	11.0	19.1	12.7	7.4	6.4	3.2
151 - 170	208	44.9	11.6	17.4	7.2	14.6	2.9	1.4
171 - 190	180	38.4	16.7	10.0	16.7	10.0	8.3	0
191 - 210	63	29	14	5	29	5	14	5
211 - 230	36	50	25	0	17	3	6	0
231 - 250	15	0	60	27	13	0	0	0

TABLE 5. *Palpable size of liver correlated with configuration of subcostal angle in 1,000 subjects*

Subcostal angle	Number of subjects	Relation of liver edge to costal margin (per cent of subjects)						
		Not palpable	At margin	1 cm	2 cm	3 cm	4 cm	5 cm
Narrow	225	55.6	11.1	14.7	9.3	5.3	4.0	0
Medium	553	41.8	13.6	14.6	11.9	9.4	7.6	1.0
Wide	222	31.5	18.5	16.2	18.9	8.1	2.7	4.1

suggested. The distributions were haphazard. Apparently healthy old people do not tend to have larger or smaller livers than healthy young people, the liver is not more or less likely to extend below the costal margin in men than it is in women, et cetera.

COMMENTS

It must be emphasized that for the clinician's purposes the configuration and consistency of the liver's edge often are considerably more helpful than its size. As previously pointed out,[3] discovery on routine physical examination that the liver extends a couple of centimeters or more below the costal margin and that, in particular, its edge is blunt and hard is the means by which many cases of chronic hepatitis are detected. In the present series of normals, the liver edge was judged to be sharp and normally firm as a criterion for inclusion in the study. It was thought that instances of liver disease were effectively excluded, both by the examiner's impression upon palpation and by the results of liver function tests and biopsy carried out in cases in which the question arose.

Findings on any routine examination are static findings, indicating only conditions as they exist at the moment. The exami-

nations recorded here were made at various times throughout the day. The normal adult liver, which weighs about 1,500 grams, contains about 800 ml of blood. The blood content, therefore, exerts an important influence over liver weight and size. Because, presumably, this blood fluctuates with the phase of digestion and physical activity, it must be supposed that liver size may vary significantly from hour to hour during the day and perhaps from day to day. This matter has not yet been explored but will be.

SUMMARY

To find figures which might be useful for the clinician in his thinking about normal limits of the liver's descent below the costal margin, observations were made on the position of the liver edge during routine physical examinations of 1,000 adults. Precautions were taken to exclude subjects with liver disease.

It was found that at the point of maximum inspiration the liver edge could not be palpated in the linea semilunaris in 42.6 per cent of the subjects, descended to but not beyond the costal margin in 14.4 per cent, descended 1 cm below the costal margin in 15.0 per cent, 2 cm in 12.9 per cent, 3 cm in 8.2 per cent, 4 cm in 5.7 per cent, and 5 cm in 1.2 per cent. No correlations could be found between the position of the liver edge and the subjects' sex, age, height, weight, or subcostal configuration.

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IMMUNIZATION REQUIREMENTS FOR TRAVELERS

Immunization Information for International Travel, Public Health Service Publication No. 384, revised to June 1958, has recently been published by the U. S. Department of Health, Education and Welfare. Copies may be obtained from the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., for 30¢ each. The booklet contains information on vaccination requirements of foreign countries and for entrance into the United States, recommended immunizations for the traveler's own protection, international vaccination certificates, and the location of yellow fever vaccination centers.

MEPROBAMATE TOXICITY

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NUMEROUS reports in the recent literature have emphasized the value of meprobamate,* a new tranquilizer, in the management of neuropsychiatric problems, particularly those associated with anxiety and tension. The agent also has been used with very encouraging results in the treatment of both acute and chronic musculospastic conditions resulting from a variety of clinical disorders among which are rheumatoid arthritis and spondylitis, fibrositis, cervical root syndrome, acute and chronic torticollis, low back muscle spasm, osteoarthritis of the hip, acute active poliomyelitis of the spinal type, and tension headaches. In addition, some enthusiasm has developed for its use in the therapy of both acute and chronic alcoholism.

Review of the published data regarding the drug's toxic effects reveals the following among the more commonly observed untoward reactions: (1) dermatological problems,[1-12] consisting of erythematous, maculopapular and vesicular eruptions in various combinations, urticaria and angioneurotic edema; (2) fever and arthralgia, usually in conjunction with the skin manifestations and frequently suggesting a serum sickness-type picture. Of less frequent occurrence are nonthrombocytopenic purpura,[13-19] sometimes seen in association with the other dermal eruptions, diarrhea, temporary paralysis of extraocular muscles with diplopia, bronchospasm, acute cerebral excitation, mild hypotension, and syncopal attacks.[3,4,11,20]

Very few fatalities have been attributed to meprobamate. A fatal case of aplastic anemia following 1.2 grams of the drug daily for a total of eight days was reported by Meyer, Heeve, and Bertscher.[21] Post-mortem findings revealed evidences of pneumonia and septicemia due to *Escherichia coli* and nonhemolytic *Staphylococcus aureus (Micrococcus pyogenes* var. *aureus)* with hemorrhages in the lungs, heart, kidney, and abdominal muscles. Powell, Mann, and Kaye[22] recently reported two cases of suicide that followed ingestion of about 12 grams and 20 grams, respectively. Most of the reports, however, have

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*Sold under the trade names Equanil (Wyeth) and Miltown (Wallace).

emphasized the relative absence of toxicity and serious side effects when the agent is used at a therapeutic level. The impression often has been created that meprobamate is an altogether safe medicament. An early study[2] reported a case where the ingestion of 20 grams of the drug in a short time produced only marked sleepiness. In discussing three patients who swallowed 6, 10, and 18 grams of meprobamate, respectively, with suicidal intention, Dixon[23] stated that all three gave the same clinical appearance of a deep, stuporous state, showing hyporeflexia and mild pupillary dilatation with poor light response. All three patients responded to painful stimulation; exhibited normal respiration, blood pressure, pulse, and oxygen exchange; and recovered fully in 4 to 6 hours, requiring only physical and caffeine stimulation. Charet, Brill, and Elloso[24] reported a suicidal attempt and mentioned four other cases about which they obtained knowledge through a personal communication with Berger. The dosage involved varied from 9.6 to 40 grams. Although the former dose produced somnolence, the 40 grams were stated to have produced no serious adverse effects. In reviewing the literature, Powell, Mann, and Kaye[22] found 11 cases of attempted suicide with large doses.

A few recent articles, however, have uncovered the drug's more dangerous potentialities. Coma associated with hypotension has been reported.[22, 24–31] In one of the patients the pulse and blood pressure could not be obtained and respiratory paralysis occurred.

Meprobromate is not believed to be habit-forming but Mohr and Mead[32] recently reported a case of apparent addiction in an alcoholic patient.

We recently attended three patients who had taken overdosages of meprobamate with suicidal intent. They are believed to be of interest not only because of the severe, prolonged coma which occurred in two with a near fatality, but also because of the striking peripheral neuropathy which developed in one of the patients.

CASE REPORTS

Case 1. A 28-year-old woman was admitted to this hospital on the morning of 8 November 1956 in a deeply comatose state. She had been under psychiatric therapy for an anxiety and character disorder, manifested by passive dependent reaction and depression. On the night of 7 November she attempted suicide by taking an overdosage of meprobamate, prescribed in conjunction with her illness, and was found unconscious in her bed the following morning and rushed to the emergency ward. After careful search of her environment and subsequent interrogation of the patient, it was determined that she had swallowed about 23.6 grams of the drug.

Physical examination on admission revealed a well-developed, well-nourished white woman who appeared deeply comatose and areflexic.

Her pulse rate was 120 per minute and thready, and her blood pressure was 74/50 mm Hg. Her pupils were widely dilated and the pupillary and corneal reflexes were absent. The pharynx and nasal passages were filled with thick secretions. Rhonchi were heard throughout both lung fields. The remainder of the examination was negative.

Laboratory data revealed an initial white blood cell count of 20,700 per μl, with 95 per cent neutrophils, 4 per cent lymphocytes, and 1 per cent monocytes. The hemoglobin was 12.7 grams per 100 ml, and the hematocrit was 40 ml per 100 ml. The sedimentation rate was normal. Four days after admission the white blood cell count had dropped to 9,100 per μl and showed a normal differential smear. The hemoglobin was 13.3 grams per 100 ml and the red blood cells numbered 4,800,000 per μl. The sedimentation rate had climbed to 48 mm per hour. Urinalysis on admission was negative but on the following day revealed a trace of albumin and 10 to 15 red blood cells and 10 to 15 white blood cells per high-power field. Blood chemistry studies the day after admission showed normal carbon dioxide combining power, creatinine, and urea nitrogen. Blood drawn on the day of admission and gastric aspirant were both negative for the presence of barbiturates. The urine was similarly negative for barbiturates and salicylates.

A dextrose-saline venoclysis containing levarterenol bitartrate was promptly started. The patient's stomach was thoroughly lavaged and aspirated—approximately 12 hours after ingestion of the drug. Aspiration of the upper respiratory passages with a suction machine and insertion of a Foley catheter also was quickly accomplished. The patient was placed in an oxygen tent and given penicillin intramuscularly.

Five hours after admission to the hospital the patient was able to maintain her blood pressure without levarterenol bitartrate, and its administration was discontinued. About 11 hours after admission, 2 ml of picrotoxin were given intravenously, followed by an additional 2 ml 20 minutes later and then by 3 ml 15 minutes afterward. A slight eyelid flutter after the last dose was the only response. Two injections of caffeine sodium benzoate were given at four-hour intervals after the picrotoxin, without noticeable effect. About 12 hours after admission the patient was still deeply comatose and areflexic and showed a gallop rhythm on auscultation. There was no evidence of hypervolemia, however, and the parenteral infusion was continued slowly. The gallop rhythm disappeared spontaneously one and one-half hours after its initial detection.

On 9 November, after approximately 24 hours of hospitalization, the patient was found to be markedly dyspneic. Examination revealed coarse rales and rhonchi throughout the entire right lung, and dullness to percussion and absent breath sounds over the left lower lung field. It was thought she had partial atelectasis of the left lung. The nasopharynx was filled with thick secretions and it was obvious that frequent suctioning throughout the night had not succeeded in maintaining an adequately clear airway. A tracheotomy was consequently

decided upon. While waiting for the necessary equipment, the patient suddenly stopped breathing and became cyanotic. An emergency tracheotomy was performed immediately and the tracheobronchial tree aspirated through the incision, removing large amounts of unusually thick and sticky secretions. The breath sounds became audible over the left lung field and the respirations and color improved rapidly. The penicillin dosage was increased and streptomycin sulfate was added to her therapeutic regimen. A roentgenogram of the chest at that time revealed an extensive pneumonic process in the right upper lung field. An electrocardiogram showed low to inverted T waves in leads II, III, aVF and V_4 through V_6 with associated depression of the S-T segment. Shortly afterward all her reflexes returned, and approximately 32 hours after admission and 45 hours after the ingestion of the meprobamate the patient began responding to stimuli and was fully conscious four hours later. Her recovery was uneventful thereafter. The tracheotomy tube was removed four days after its insertion. The lung fields became clear and the electrocardiographic tracings reverted to normal. The patient was discharged in good physical condition and psychotherapy was continued on an outpatient basis with excellent results.

Case 2. A 19-year-old enlisted man with previous excellent record of conduct and good health was being seen at the mental hygiene clinic of this hospital because of a recent onset of bitemporal headaches and extreme nervousness and anxiety, manifested by insomnia and feelings of compulsion to vigorous motor activity. A prescription for meprobamate with usual dosage schedule was written on 7 September 1956. The youth was found unconscious on the floor of his barracks in the early morning of 11 September with a suicidal note beside him. There also was evidence of considerable vomiting, and two empty boxes of medicine were nearby. It was believed, after tracing the prescribed medications and subsequent interrogation of the patient, that he had taken 20 grams of meprobamate and an undetermined, though seemingly not dangerously excessive number of acetylsalicylic acid tablets.

The patient was rushed to the hospital where initial examination revealed a well-developed, slender, deeply comatose youth with a regular pulse of 140, a blood pressure of 120/70 mm Hg, and a respiratory rate of 32 per minute. His pupils were widely dilated and fixed, and his corneal and all deep reflexes were absent.

Admission laboratory data was as follows: white blood cell count, 16,000 per μl, with 81 per cent neutrophils and 19 per cent lymphocytes; hemoglobin, 15.6 grams per 100 ml; and hematocrit 48 ml per 100 ml. Two urinalyses showed 1 and 2 plus albuminuria. Microscopic examination of the urine revealed 20 to 25 red blood cells per high-power field on both occasions, but was otherwise negative. A urine test was negative for barbiturates but positive for salicylates (145 mg per 100 ml). Blood chemistry studies revealed a blood urea nitrogen of 16.2 mg per 100 ml and carbon dioxide combining power of 17.7 mEq per liter.

The patient was adequately lavaged and aspirated, placed in an oxygen tent and given 16 ml of picrotoxin intravenously without noticeable response. A spinal fluid examination was normal. Around 0700 hours on the following morning (12 September) he was still deeply comatose and areflexic, and his blood pressure dropped to alarming hypotensive levels. The urinary output had been nihil and he had been running a continuous fever since shortly after admission. Levarterenol bitartrate was added to the intravenous fluid regimen, with excellent blood pressure response. Rhonchi were heard at that time in both lower lung fields and a roentgenogram of the chest showed diffuse infiltrative lesions in both lung fields, particularly on the right. Fearing an aspiration pneumonia, he was started on penicillin, intramuscularly, and streptomycin sulfate in the accepted dosage. His carbon dioxide combining power was 20 mEq per liter and serum chlorides were 114 mEq per liter. Around 1030 hours on 13 September he was able to maintain his blood pressure spontaneously, and levarterenol bitartrate was discontinued. Normal, equal tendon reflexes were elicited at that time. Around 1300 hours of the same day the first traces of conscious behavior were detected, and by 1630 hours in the afternoon he was fully conscious. The oxygen therapy was discontinued. His urinary output improved rapidly. The urinary abnormality, *i. e.*, albuminuria and microscopic hematuria, persisted until 14 September, after which it disappeared. His hemogram was now within normal limits. An electrocardiogram was interpreted as normal. His fever, however, continued unabated. He developed a fine maculopapular and erythematous eruption on 16 September and typical urticarial lesions on 18 September, both of which disappeared during the following week.

The only physical findings of significance at the time of his hospitalization, outside of his continuous febrile curve, were coarse rales and rhonchi throughout both lung fields, particularly at the bases. His white blood cell count, differential smear, platelet and red blood counts were normal, and his sedimentation rate was 21 mm per hour. A chest film on 18 September showed persistence of the diffuse infiltrations in both lung fields indicative of bronchopneumonic processes. In view of the dermal lesions and persistent fever, it was decided to change antibiotics, and Terramycin (brand of oxytetracycline) was accordingly started while discontinuing the others. Toward the end of the second week, the patient developed considerable respiratory difficulty, became cyanotic and delirious, and his cough and purulent expectoration increased. He maintained his continuous febrile course with an average oral temperature of 103°F. Physical signs of dullness to percussion, decreased breath and voice sounds, and crepitant rales were noted over the right anterior lung field at the level of the 3d to 5th rib for about 4 cm lateral to the sternum. A repeat chest film revealed a new infiltrative lesion in the outlined area. A leukocytosis with neutrophilic predominance and a markedly elevated sedimentation rate were now present. It was evident we were dealing with a severe pulmonary infection obviously unresponsive to the antibiotics the patient had received. Oxygen therapy was re-

started. More intensive study of the patient's sputum showed a florid, almost pure culture of *Micrococcus pyogenes* var. *aureus*, coagulase-positive on three occasions with good erythromycin sensitivity. This antibiotic was consequently started in large doses on 26 September after Terramycin had been discontinued. Several blood cultures were negative. The patient's temperature curve exhibited a dramatic response, with his fever beginning to subside on 28 September, reaching normal levels quickly, and remaining flat thereafter. This coincided with a fairly prompt disappearance of his associated symptomatology and physical findings, and radiographic resolution of his pulmonary process. A few cystic areas remained in the region of the right middle lobe for several months.

About 28 September the patient started complaining of pain in the ball of his right foot, and a few days later of soreness in the right calf and posterior thigh muscles. At that time he had normal tendon reflexes and tenderness to palpation over that musculature without swelling, heat, or discoloration. His right foot pain rapidly became more severe, and hyperesthesia developed to the point where even contact with the sheet would produce an excruciating discomfort which was described as a burning sensation. Examination then revealed considerable right sciatic pain on straight leg raising, a right foot drop, and disappearance of his corresponding Achilles tendon reflex. Coughing caused severe pain in his right foot. Some sensory loss was detected over the posterior aspect of his right thigh. In view of the sequence of events, the lack of a history of back trauma, the absence of tenderness to pressure over the back region, and the negative roentgenographic findings of lumbosacral area, it was believed we were dealing with an acute neuritis of the right leg rather than spinal root involvement. The patient was given two courses of massive vitamin B_{12} therapy, consisting of 1,000 micrograms of Rubramin intramuscularly every day for 10 days with a week's rest period. He was also promptly started and maintained on physiotherapy for about two months. His discomfort gradually subsided over a period of two weeks and he showed considerable, though gradual, improvement in his peroneal paralysis. He was evacuated to Valley Forge Army Hospital, where both psychotherapy and physiotherapy were continued with further over-all improvement in his condition, and he was discharged to duty with minimal assignment limitations.

Comments. The role played by the ingestion of salicylates in the initiation and maintenance of this patient's comatose state deserves careful scrutiny. The patient claimed he had consumed a considerable number of the prescribed acetylsalicylic acid tablets in a normal schedule prior to his suicidal attempt. He was unable to state the size of the original prescription. The dimensions of the box which had contained the tablets, however, indicated a maximum capacity of about 20. Furthermore, the evidence of his having vomited suggested the rejection of a good portion of the drug. The urinary salicylate determination of 145 mg per 100 ml may, at first glance, seem like a

relatively high figure. It is known that about 70 to 80 per cent of ingested salicylates are excreted by the kidneys and that the percentage excreted bears no precise relationship to the dose administered or the urinary volume. Consequently, the urinary level after a fixed dose may vary considerably, depending on the urine specific gravity, appearing unusually high in concentrated urine and low in diluted urine. The patient's impaired urinary output during the first 24 hours following his suicidal attempt must have contributed greatly to the urine salicylate level obtained. Unfortunately, we have no data on his blood level, which is a more accurate and reliable criterion of the degree of salicylate intoxication. There is actually no known correlation between blood and urine salicylate levels where the urine salicylate concentration is expressed as milligrams per 100 ml in a random sample.

It is believed that this patient's clinical picture was not indicative of significant salicylate intoxication. Tinnitus and auditory nerve involvement are frequent manifestations of moderate or severe salicylate poisoning. These were conspicuously absent in our patient. A disturbance in acid-base balance is a constant finding in salicylate poisoning. This is particularly pronounced in the more severe intoxication, a situation that would exist were a prolonged comatose state etiologically related to salicylate overdosage. The blood chemical data in the early stages of the intoxication are characterized by low carbon dioxide levels and serum pH values in the high normal or alkaline range, or respiratory alkalosis. This abnormality is thought to be a result of hyperventilation, mainly hyperpnea due to a specific action of salicylate in stimulating the respiratory nervous mechanism. In more severe poisoning this initial respiratory alkalosis is followed by a true metabolic acidosis with primary carbon dioxide and alkali deficit, marked reduction of buffer base and a shift of the serum pH from the alkaline to the acid side of normal. This later phase acidosis, incidentally, is most probably caused by organic acid metabolites[33] arising from changes in the intermediary cellular metabolism resulting from the action of salicylates rather than the salicylate radical itself. Ketosis with acetonuria is commonly encountered at this stage. It is obvious that our patient did not exhibit a comparable clinical picture. His initial hyperventilation was more in the nature of tachypnea rather than hyperpnea and its duration was short. It may be claimed that meprobamate might have interfered or modified this characteristic respiratory manifestation. No serum pH studies were made. His blood carbon dioxide was somewhat low on the day of admission (17.7 mEq per liter). However, this returned to normal levels during the following 24 hours, despite the fact that he did not receive any alkaline fluids parenterally, and remained so thereafter. This bespeaks

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rather convincingly against the development of any significant degree of metabolic acidosis.

Case 3. A 23-year-old enlisted man with a severe emotional instability reaction received a prescription for 25 meprobamate tablets (total of 10 grams) on 14 November 1956 as part of his therapeutic regimen. On 15 November, at about 0500 hours, he swallowed all 25 tablets, ate breakfast, and reported for an appointment at the clinic at 0800 hours. He appeared confused and in an obvious state of drug intoxication. He admitted taking the overdosage of the drug, and was rushed to the hospital's emergency room. His stomach was immediately aspirated and lavaged—approximately four hours after swallowing the meprobamate.

Physical examination on the ward revealed the patient to be a slender, well-developed, Caucasian who was markedly lethargic but could be aroused. His pulse rate was 96 per minute and regular, and his blood pressure was 100/80 mm Hg. Except for the lethargy and generalized muscular weakness, the physical examination was otherwise negative.

The laboratory data were as follows: white blood cell count, differential smear, hemoglobin, hematocrit, and urinalysis were all normal. Sedimentation rate was 12 mm per hour. The urine and blood samples on admission were negative for the presence of barbiturates. A roentgenogram of the chest was interpreted as normal.

The patient was placed under close observation and given 5 mg of Dexedrine Sulfate (brand of dextro amphetamine sulfate) orally every four hours. He improved rapidly and within approximately five hours after entering the hospital, and 11 hours after taking the overdosage of the drug he was alert and cooperative. After 24 hours the Dexedrine dose was reduced, and after 48 hours was discontinued. The patient was discharged from the hospital in good physical condition five days after admission.

COMA AND HYPOTENSION

The precise metabolic mechanisms involved in the production of coma and hypotension by meprobamate have not been elucidated.[34] In animal experiments,[35] massive doses of meprobamate produce a reversible flaccid paralysis of most voluntary muscles and thereby resembles curare in its action, except that the respiratory muscles are invariably spared. This is the picture usually encountered in comatose patients who have taken suicidal overdoses. Hiestand[29] postulated that the loss of muscle tonus somehow interferes with the ability of the vascular system to maintain normal tone with consequent drop in pressure. It would be interesting in this connection to correlate the degree of muscle relaxation and deep tendon reflex depression with the presence or absence and severity of hypotension. Another mechanism, however, would have to be sought to explain the hypotensive phenomenon described after ingestion of one tablet

of meprobamate with the patient remaining fully conscious and ambulatory. A direct action on the vasomotor center of the medulla is, of course, another possible explanation.[29]

An individual susceptibility to meprobamate is suggested by the wide variations in the dose producing serious degrees of central nervous system depression among different patients. Whereas 40 grams have been reported to have produced no serious side effects,[24] Allen and Black's[28] patient became pulseless and developed respiratory paralysis after ingestion of 12 to 16 grams of the drug, despite her early gastric lavage and probable removal of a certain amount of the agent. Also in marked contrast to the latter case, our last patient merely manifested a deep slumber and was easily aroused at all times although he ingested 10 grams of meprobamate and was lavaged quite late. Dixon's[23] patients revealed identical clinical pictures, yet one took 6 grams while another swallowed 15 grams of the drug. Hiestand's[29] patient apparently never was in serious distress and showed a return of pupillary reflexes in 4 to 6 hours and good muscular response 18 hours after taking 36 to 38 grams of meprobamate. Yet, of our own first two patients, one was a near fatality and the other suffered a severe 48-hour coma with prolonged hypotension after taking 23.8 and 20 grams respectively. Statements regarding the amount of drug swallowed may be subject to question, particularly in view of the personality of the patients involved.

PERIPHERAL NEUROPATHY

The peripheral neuropathy which developed in our second patient is a subject for interesting speculation. Careful review of the literature failed to reveal a single instance of peripheral nerve involvement following the use of oxytetracycline, erythromycin, or picrotoxin, agents used in the patient's therapeutic management. Similarly, there is no published report of a staphylococcal infection or salicylate intoxication producing such a morbidity. Streptomycin sulfate, also received by the patient, is well known for its neurotoxicity. It frequently involves the eighth, less commonly the third and second cranial nerves, and in its incipient years occasionally produced a fatal encephalopathy when extremely large doses were being given. However, as with the preceding agents, there is no official reference implicating the antibiotic in the causation of such a neuropathy. In 1946 Kolb and Gray[36] reported seven cases of peripheral neuritis following repeated intramuscular injections of penicillin for various infections. The onset of the neuritis was observed between 10 and 21 days after the institution of penicillin therapy and, in some cases, while receiving the antibiotic. The protocol of four of the seven cases mentions the injection of penicillin in the area of a nerve trunk later involved by the neuritic process. In the others there is no description

of the injection site. In 1949 Broadbent, Odom, and Woodhall[37] described four cases of peripheral neuritis following intramuscular penicillin administration convincingly shown to be due to nerve injury from the local drug infiltration. Several of those accidents may well have been related to impurities present in the penicillin preparations available at that time. Considering the number of years that penicillin has been in the world market and its widespread popularity, the reporting of so few cases suggests that this complication following its use must indeed be a rarity. Nevertheless, it is a possibility which should be entertained were a peripheral neuropathy to develop during penicillin therapy. It is difficult, however, to escape the conclusion that meprobamate could be implicated in its causation. The drug certainly can produce profound, though as far as it is known, reversible alteration in muscle tonicity and behavior through its action on intranuncial nerve fibers. The observation is being offered for its factual value and as a reference for future elucidation.

THERAPEUTIC CONSIDERATIONS

In animal experiments[34] Metrazol (brand of pentylenetetrazol) was found to be the most effective antidote to meprobamate, producing a definite shortening of the comatose state. Picrotoxin proved quite ineffective. This adds credence to Heberden and Cooper's[31] description of their patient's dramatic response to intravenous metrazol, becoming fully awake within the ensuing 60 seconds after having been in coma for many hours following ingestion of slightly over 8 grams of meprobamate. It also explains our two unsuccessful experiences with picrotoxin.

The associated hypotension has shown consistent prompt response to intravenous levarterenol bitartrate, though in a few instances its administration had to be continued for many hours. Allen and Black[28] kept their pulseless patient with respiratory paralysis alive by using, besides levarterenol, cerebral electrostimulation with a Med-Craft Electronic Transient Therapy Unit. This was applied over a period of three hours before the patient was able to maintain her own respiratory activity. No Metrazol was used however.

It is obvious that careful evaluation and prompt reversal of all serious threats to life as they develop should be an integral part of the patient's management and need no further comment.

There are now available a qualitative urine test[38] for meprobamate and a colorimetric procedure[39] for the presence of the drug in body fluids where a question of diagnosis exists in instances of coma of unknown cause.

SUMMARY AND CONCLUSIONS

Meprobamate has gained rapid acceptance among members of the medical profession as an effective agent in the manage-

ment of musculospastic disorders and neuropsychiatric problems, particularly those associated with anxiety and tension. Its administration has in certain instances, however, led to some serious, even fatal, reactions and uncovered its dangerous potentiality when used without judicious discernment. This facet of its toxicity was analyzed and discussed after reviewing reported instances of severe reactions with particular reference to coma and hypotension. Consideration was given to a few valuable therapeutic observations. Experiences with three patients who took overdoses of meprobamate with suicidal intention were presented and added to the discussion. The importance of exercising good judgment in prescribing the drug, especially to depressed, potentially suicidal or seriously disturbed individuals, was strongly emphasized.

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TUBERCULIN TESTING OF MILITARY PERSONNEL

Tuberculin testing, now well established by the Navy, is ideally suited to the needs of the armed forces as a tool for detection of tuberculous infection. This is particularly important because 90-95 per cent of recruits enter service with negative skin tests. Their duties carry them occasionally to parts of the world where opportunity for infection is great. Such infections should be service connected, even if only identified by a conversion of the tuberculin test. Possibly they should be treated at this stage—perhaps the armed forces could determine for us, by means of a well designed study, if they need to be treated.—H. Corwin Hinshaw: Speculations on the Future Treatment and Control of Tuberculosis. *Diseases of the Chest,* September 1958.

MEDICAL RADIATION AND COMMON SENSE

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SHORTLY after Röntgen announced the discovery of x-rays in November 1895, it became obvious that this strange new energy possessed some hazardous properties. By 1898, the British Roentgen Society was already collecting information "on the injurious effects of roentgen rays."[1] Knowledge of the extent of these hazards and the degree of protection required was slowly accumulated in the years 1900-1935. By 1935 the collection of such data had been tremendously accelerated by the international acceptance in 1928 of a unit of measurement of ionizing radiation, the roentgen, and by improved instrumentation permitting accurate mensuration of these units.[2]

Fortunately, awareness that the rays were potentially harmful did not prevent the pioneers in medical radiology from developing this field as a diagnostic and therapeutic discipline. The rapid advances and developments in radiology in less than threescore years have been awe-inspiring, and the changes wrought upon the entire field of medicine by utilization of ionizing radiation are of immeasurable importance. For instance, Coller[3] has pointed out that, prior to the discovery of roentgen rays, surgical operations were limited to conditions that Hippocrates could have diagnosed easily.

Nearly sixty years later, a rather sudden popularization of the uses and hazards of radiation through the medium of the lay press has become a problem of great importance and concern to medical and dental practitioners. We now find ourselves treating a patient population that is confused, apprehensive, misinformed and reluctant with regard to the medical uses of x-ray and radiant material. The public has been exposed to a plethora of articles by the press—some well-intentioned but lacking clarity, others frankly terrifying in their approach and conclusions.

Consequently, a new medical problem has arisen: the patient so resistant to the use of diagnostic and therapeutic radiation that he wishes to forego it and revert to more primitive modalities. We have seen a strong man, a former military pilot, turn

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pale and tremble when told he would have several hundred roentgens delivered to a pituitary tumor. He had read that a dose of 600 r from atomic weapons produced immediate death. As a world-renowned radiation physicist has put it, we have now reached the point where "the hazards of dealing with the hazards are greater than the hazards."[4]

A calm reappraisal of the hazards, advantages, protection, and uses of diagnostic ionizing radiation in medicine seems in order. However, consideration of the problem of radiation effects upon the population can not and should not be limited to radiant sources utilized in medical and dental practice.

SOURCES OF RADIATION

The population of the earth is and has been constantly bombarded with ionizing radiation. The sources of this radiation include cosmic rays, naturally occurring radioactive elements, man-made radioisotopes, medical and dental x-ray generators, nuclear fall-out from atomic weapons, and industrial generators and reactors.

The major portion of background radiation (from cosmic rays, radioactive rocks and soil, and internal radiation from radioactive components of the body) cannot be effectively controlled but is variable under different circumstances. For instance, the 30-year accumulated, gonadal dose of a person living at sea level in a frame house is 3 r or more. If this person moved to a similar house in Denver, the cosmic-ray activity at this altitude would increase his 30-year gonadal dose by 17 per cent. Moving to a brick, concrete, or stone house in Denver would increase his dose another 33 per cent. Living on the sandy beaches of Travancore, India might produce a 30-year accumulation as high as 50 r.[5] So far, an increased incidence of radiation injuries related to brick houses in Denver or mud huts in Travancore has not been described.

The gonadal dose to the people of Great Britain from diagnostic x-rays is reported to be only 22 per cent of that received from natural sources.[4]

DELETERIOUS EFFECTS OF RADIATION

The harmful effects of ionizing radiation upon the body as listed by The International Commission on Radiological Protection[6] are:

1. Superficial injuries.
2. General effects on the body, particularly the blood and blood-forming organs, e. g., production of anemia and leukemias.
3. Induction of malignant tumors.
4. Other deleterious effects, including cataract, obesity, impaired fertility, and reduction of life span.
5. Genetic effects.

Some knowledge of these effects will facilitate clarification of patients' misconceptions regarding somatic injuries.

Superficial Injuries. Acute superficial injury of the skin was first noted within one month of Röntgen's discovery.[7] Subsequently, chronic effects became apparent after a latent period of varying length. Because of lack of data on the dose received by most individuals injured in the past, it is difficult to form a concept of permissible dose which precludes injury.[1] Doses in the order of 4 r per week have produced skin changes.[8] The safe tolerance dose has been given as 0.5 r a week to the surface of the body. This dose corresponds to 0.3 r a week measured in free air.[6] It should be pointed out that the effects of overexposure of skin are, to a large extent, independent of the size of the area exposed. This is in contrast to other tissues, such as the hematopoietic system, where destruction or injury to one area may be compensated for by proliferation in another area.

Acute superficial changes, erythema and epilation, require large doses in the order of 250-1,000 r depending on the wave length. Chronic changes which include atrophy, telangiectasia, radiodermatitis, chronic ulceration and carcinoma require large doses over an extended period of time. With present-day knowledge and equipment, these injuries should not constitute a problem in diagnostic radiology.

Effects on the Blood and Hematopoietic Tissue. Since 1903, it has been known that radiation affects the blood and blood-forming organs. There is wide variation, both qualitatively and quantitatively, as to the effect on particular blood elements according to the amount of radiation, the quality and source of the radiation, the conditions under which irradiated, and the observer reporting.

Aplastic anemia has occurred in some radiologists who apparently received large doses.[1] It may be produced in guinea pigs with exposures of 2.2 r per day.[9,10]

Leukemogenic effects of radiation have been widely publicized. Evidence that there is a correlation between exposure to ionizing radiation and development of leukemia is overwhelming. March[11] observed that leukemia is nine times as frequent in radiologists as in other physicians. Henshaw and Hawkins[12] have reported that the incidence of leukemia in physicians is almost twice that in the population as a whole, and that this may be because of the use of and exposure to x-rays and radium. Leukemia may be induced experimentally in irradiated animals.[1] Leukemogenic effects have been described in survivors of the Nagasaki and Hiroshima explosions and in British children irradiated as fetuses.[13,14] Quantitative data as to what constitutes a leukemogenic dose is lacking and no patient can honestly be told that such and such dose will increase his risk of leu-

kemia. Experimentally, a given dose of partial body irradiation will not increase the normal incidence of leukemia in mice, but when given to the entire body, the incidence is increased.[15] Diagnostic radiology is practically based on partial body exposure.

The literature is replete with experimental and clinical cases of radiation-induced malignancy. Besides leukemia, there are reports of skin cancers in previously treated areas,[16] thyroid carcinoma following irradiation of the thymus during infancy,[17] and of various tumors experimentally produced in animals.[1] These occur at dosage levels which should not be encountered in diagnostic procedures or current methods of radiation therapy.

Other Deleterious Effects. Most widely known and oft-quoted of these "other deleterious effects" are reduction of life span and impaired fertility.

Information on life shortening by irradiation is derived mainly from animal experiments utilizing high dose rates or large doses.[18] In man, reports[19-21] have indicated that the life span of radiologists is shorter than that of other physicians or other medical specialists. This has been attributed to chronic radiation exposure. Recent reappraisal[22] of these figures, however, does not support this hypothesis and suggests that the difference is related to differences in age composition of the groups. There is no direct method of relating life span to past radiation exposure.[23] Many radiologists dying in earlier groups were older men who may have received 75 times the dose encountered by present-day radiologists with modern equipment.

Impaired fertility or sterility can be produced by large acute doses of radiation. Irradiation of 625 r delivered in a few days to the ovaries may produce permanent amenorrhea, and slightly higher doses to the testes, aspermia. However, a complete gastrointestinal series yearly for 30 years would not produce sterility dosage levels.[5] Impaired fertility or reduction in the number of offspring as a result of radiation exposure is almost impossible to evaluate in our complex society.

Genetic Effects. The effect of ionizing radiation on the normally unchanging gene has been the prime object of much recently gained public attention. Naturally occurring gene change or mutation is extremely rare, but in all species thus far studied, it has been found undesirable.[24] It is accepted that radiant energy, along with chemicals, fever and other factors, increases the mutation rate. Moreover, there is no known lower limit of gonadal exposure at which change does not occur. Thus, in order to gain a reference point, an estimated dose that will double the mutation rate is used, called "the doubling dose." The Genetics Committee of the National Academy of Sciences has taken the range of 40 to 80 r to the human gonads as "the doubling dose." Later calculations by Glass[25] suggest a gonadal

dose as low as 10 r may double the mutation rate. Carrying these assumptions further, a doubling dose delivered to the gonads of the country's entire reproductive-age population would likely increase the present rate of genetic defects among newborns from 2 to 4 per cent.

When discussing genetic effects, it must be kept in mind that it is not possible to avoid radiation from cosmic sources and the earth. Furthermore, a given individual's dose is not the important factor but, genetically, it is the *average* to the *entire* population between conception and the end of child-bearing that is critical. Diagnostic radiation to older people is of no consequence.[26]

Somatic vs. Genetic Effects. The difference between somatic and possible genetic damage has not been made clear by recent publicity. This lack of clarity and completeness has undoubtedly enhanced the fears, mainly unfounded, that are prevalent in the public and not a few physicians. When discussing this subject with patients, one soon observes that the most alarming misconception is an erroneous association of small genetic dosages with personal somatic harm. This strong but misguided motivation for self-preservation occasionally is manifested by a patient refusing to submit to a routine chest film. Yet, this same person has no hesitancy about wearing a luminous dial watch which may contribute an even greater dosage.

One must constantly bear in mind that the low dosages of radiation which the geneticists suspect may be harmful refer to gonadal dosage to the *entire* population. Many diagnostic procedures are adequately performed without measurable radiation reaching the gonads. Further, this amount of radiation has not been shown, experimentally or otherwise, to induce somatic damage to the individual.

Reassurance of an individual who is apprehensive regarding radiographic procedures is most effective when it is made clear that he or she as an individual is in no danger of harm. Most incompletely informed patients we encounter are anxious about their own health rather than that of future generations. Quimby has accurately described the present state of mind created by incomplete or inaccurate lay articles, "They (the public) do not know what they are afraid of—they are just afraid."[26]

The physician armed with a clear understanding of the difference between somatic and genetic hazards and with a few comparative figures can ease many patients' fears. Hodges[5] states that evaluation of available data indicates that, with the possible exception of situations in which the gonads must be in the direct beam, diagnostic radiology conducted by qualified radiologists with proper equipment contributes an insignificant part of the total gonadal irradiation over a 30-year period when compared to existing background radiation. As far as the danger of somatic harm is concerned, the best current estimate[27]

regarding total whole-body dosage from *all* sources received annually by the average U. S. citizen is 0.3-0.4 roentgen absorbed. Many times the dosage dealt with in diagnostic radiology when delivered over a period of many years has not been shown to cause detectable individual damage.[26]

THE MAXIMUM PERMISSIBLE DOSE

The maximum permissible dose applies to those individuals who are occupationally exposed to ionizing radiation. Details of the derivation and exact definition of this phrase are to be found in radiation protection handbooks of the National Bureau of Standards.[29] All present values and concepts in use today regarding maximum permissible dose result from recommendations of the National Committee on Radiation Protection and the International Commission on Radiological Protection.[30] There are many facets to this subject and for the physician a working concept is most valuable. At present, the rule is that protective barriers and measures should be such as to limit radiation to workers to a maximum of 0.3 roentgens in air whole-body dose per week or 210 roentgens accumulated dose by age 60.

The concept of maximum permissible dose should be applied with the understanding that the expressed limits are merely an educated guess as to the dose of ionizing radiation that, in light of present knowledge, is not expected to cause detectable bodily injury to a person at any time during his lifetime. The maximal permissible dose does not represent a tolerance limit. In a practice which is properly regulated and monitored, it is found that radiologic personnel usually receive much less than 0.3 r per week. In our own department, servicing a 900-bed hospital, the average exposure to radiology personnel is less than 10 per cent of the maximum permissible dose.

Committees responsible for radiation protection handbooks stipulate that radiation exposure resulting from necessary medical and dental procedures shall be assumed to have no effect on the radiation exposure or tolerance status of the person concerned. This stand is based on the conviction that it is unwise to assume quantitative information when none exists.[5]

Natural radiation sources contribute in the neighborhood of 0.015 r per week or one-twentieth of the maximum permissible level of 0.3 r. Chamberlain[2] described most radiations workers as receiving about 0.03 r per week, an amount which he concludes does not seem to be an excessive increase over the natural background. A properly performed "routine" chest film gives an absorbed partial body exposure of approximately 0.05 r and less than 0.001 r to the gonads.[27] It is apparent that the maximum permissible dose of 0.3 r and a working actual exposure in the order of 0.03 r leaves considerable leeway for performing necessary radiographic procedures on those people who are occupationally exposed to radiation. Before being subjected to diagnostic or survey radiographic procedures, the

increasing number of occupationally exposed individuals in industry, military and medical fields will place an increasing demand upon the physician to explain the concept of maximum permissible dose in similar terms.

RADIATION PROTECTION

The significance of radiation protection is drilled into the radiology resident during his entire specialty training period. For many years the American Board of Radiology has required applicants to be well informed in all aspects of protection. As only 4,000 of the approximately 126,000 professional users of x-ray equipment have comprehensive, special training in radiology,[31] a brief discussion of protection is presented here.

Medical x-ray protection is directed toward elimination of all unnecessary radiation to the physician, technician, and auxilliary personnel as well as to the patient. The population may be divided into four groups, each with different problems within the sphere of avoiding all unnecessary radiation:

Group I: Those individuals whose occupations entail exposure to radiation. As mentioned earlier, this group is increasing in number. Available evidence indicates that present dosage limits are adequate to avoid significant somatic effects.

Group II: Those with illness (including the pregnant mother and fetus) at any age from conception to the end of the reproductive period. It is in this group that potential genetic hazards are greatest and that extra protective measures are most worthwhile.

Group III: Those with illness beyond the reproductive period. This includes most people over 30 years of age and practically all people of 40 years or more. Genetic hazard is no longer a problem. With rare exception, as stated earlier, there is no risk of somatic damage to these individuals in diagnostic radiology. Fortunately, most patients requiring extensive diagnostic studies fall into this category.

Group IV: The well individual. Medically, these people should receive the same consideration as the reproductive-age group regardless of age. This is in keeping with the principle of avoiding all unnecessary radiation. This does not imply that "early detection" procedures are to be avoided. For example, a properly performed chest film (fig. 1) delivers no measurable radiation to the gonads.

Obviously, it is desirable that radiation exposure to individuals in each of these four groups be held to the minimum dose *consistent with their medical requirements.* It is important to realize that the amount of radiation consistent with medical requirements is different for each group. Furthermore, any given individual may, during his life span, fall into several or all of these groups.

Recommendations that a permanent record or "radiation passport" be maintained for everyone, with every exposure noted,

are impractical for a number of reasons.[5,32] First, people are prone to lose records when individually responsible and an elaborate cross-filing system is expensive and bulky. Second, the average x-ray department cannot accurately estimate doses delivered to various body parts by different examinations. Third, accurate quantitative knowledge for the threshold doses for most injurious effects is not available. Finally, such a "passport" would further intimidate a public already wary of medical x-ray procedures. This does not imply that it is not feasible to monitor personnel occupationally exposed to radiation. On the contrary, monitoring and dosage recording on these individuals is a worthwhile necessity.

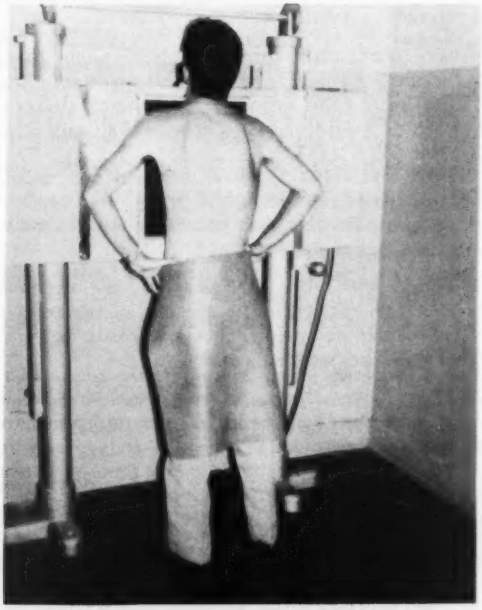

Figure 1. Lead-rubber shield with adjustable belt made from old fluoroscopy apron for use in chest filming.

A logical approach to the problem of protection from medical x-ray is for it to be handled by the physicians involved. This is the approach utilized in all similar medical problems.

How can radiation exposure be reduced to patients? There are five prime areas of consideration: (1) improved technics, (2) improved equipment, (3) special devices, (4) careful selection of examinations requested, and (5) careful selection of patients examined.

Each one of these areas is the responsibility of the physician or physicians involved for implementation. Methods by which the physician can reduce radiation exposure to the population will be considered singly and in some detail.

Improved Technics. The physician who studies and applies the latest technics is doubly rewarded with improved diagnostic results as well as greatly reduced patient and staff radiation dosage.

Higher voltages, with rare exception, do not significantly alter the diagnostic quality of radiographs. This increases penetration of the beam, while decreasing the total quanta of rays that must strike the patient to produce a satisfactory film or fluoroscopic image. Resulting films may demonstrate less contrast or eye-appeal, but reduction in exposure is considerable.

Dark adaptation is emphasized in every discussion of fluoroscopy. Importance of this expedient warrants its mention again. At least 20 minutes in darkness are required to achieve complete adaptation. When red goggles are worn for this period of time in light surroundings, an additional 2 to 3 minutes in a dark room are necessary to bring adaptation to maximum. When properly dark-adapted, the fluoroscopic procedure is performed with a smaller amount of radiation while achieving the desired results in less exposure time.

Where possible, one must utilize the decrease in exposure that results from application of the inverse square law, i. e., intensity of radiation decreases in relation to the square of the distance. In radiography this means using a minimum of 36 inches target-film distance. In fluoroscopy, this means one must require tables of a design that permit a minimum of 14 inches from the tube target to table top; 18 inches is even more desirable and should be demanded of future equipment.

Irradiating the smallest area consistent with desired results not only decreases patient exposure, but greatly enhances the fluoroscopic image and quality of radiographs by decreasing scatter or stray radiation. This is achieved by cones, shutters or diaphragms. Diaphragms must be constructed to limit the fluoroscopic beam so as to leave one-half inch of inactivated fluoroscopic screen of all margins with the screen at maximum distance above the table top and with the shutters fully open.

Addition of filtration in the primary beam has little effect on resulting fluoroscopic images or roentgenograms but greatly reduces patient exposure. Filtration removes a large portion of long wave length or "soft" radiation that is absorbed by the patient and does not reach the film or fluoroscopic screen to serve a useful purpose. The resulting beam is made of fewer but more penetrating short wave length or "hard" rays. Addition of a 3-mm aluminum filter to inherent tube filtration reduces the skin dose by 80-85 per cent in ranges up to a 130 peak kilovoltage (kvp).[33]

The need for 3 mm of added aluminum filtration in radiographic and fluoroscopic beams is made obvious by reductions of this magnitude.

Significance of x-ray protective surveys cannot be overemphasized, although the scope of this discussion does not permit detailed elaboration. It should be pointed out that such a survey will provide the physician with knowledge as to the output of the fluoroscopic beam at the table top. This output should be not more than 10 r per minute in air. In actual practice we have found that modern equipment provides a satisfactory image using 3-mm aluminum added filtration, factors of 3.5 milliamperes (ma) and 93 kvp yielding a beam of 4-5 r per minute in air at the table top. For infants and small children, factors of 2 ma and 60 kvp reduce this output to 1.5-2.0 r per minute.

A complete discussion of the physician's own fluoroscopic technic in regard to patient exposure will not be attempted. The use of these technics together with the many refinements discussed in texts of radiology require study and conscious application until proper economy of action and habit patterns are achieved. Macht and Kutz[34] demonstrated how experience of the radiologist is a major factor in reducing the time required to perform a fluoroscopic procedure. The average fluoroscopic exposure time required by a third-year radiology resident to perform a gastrointestinal examination was 5 minutes and 40 seconds; an experienced radiologist required an average of 3 minutes and 19 seconds. This time difference alone gives an approximate 30 per cent decrease in patient exposure.

Leaded rubber gloves and lead-rubber or lead-glass aprons must be routinely used during fluoroscopy. Relatives or hospital personnel not associated with the radiology department should be similarly equipped when it is necessary for them to hold or support a patient during examination. Radiologic personnel should never be utilized for this purpose.

Improved Equipment. Manufacturers must balance economy against new innovations or improvements on equipment. The physician must require modern equipment and be willing to accept the additional cost. High capacity equipment is necessary to achieve high-voltage radiology, short exposure times and consistent results under all conditions. The various government medical services provide comprehensive specifications to assure initial procurement of proper equipment. It is the physician's responsibility to provide the stimulus for modification from time to time as additional improvements become known or available.

Image amplifiers offer considerable possibility in decreasing fluoroscopic exposure and are beginning to come into clinical use. A weak fluoroscopic beam is used to create a stimulus which is passed through a series of photomultiplier tubes to

produce an image so bright that dark adaptation is not required. Hodges[5] evaluates amplifiers as being useful in examining thin parts but states that currently available amplifiers have only limited usefulness in thick parts such as the abdomen.

Radiation exposures can be sharply reduced by the use of fast films, fast screens, and correct film processing. Correct film processing requires that optimum exposure time be consistently obtained and that the darkroom use exact time and temperature developing with rapidly acting fresh solutions.

Special Devices. There are any number of opportunities to improvise or improve upon shielding methods. Application of shielding requires interest and desire on the part of the physician. The technician staff will reflect the physician's interest by using and devising shielding in every possible situation.

Shielding is most useful when protecting the gonads, but can be applied to larger areas when repeated examinations are required. Shielding must be modified to satisfy diagnostic needs but can often be applied to a portion of an examination, i. e., omit shielding for pyelograms on survey and erect films and use on all other exposures in the series. Shielding is useful in examinations requiring multiple exposures and when the primary beam cannot be directed away from the gonads, particularly in children and pregnant women. Shielding may reduce gonadal exposure during fluoroscopy to a slight extent but has little practical value in properly executed chest radiography of the adult other than as a form of reassurance. It is most worthwhile to shield for chest radiography of infants and children since the distance between the area of study and the gonads is small.

Careful collimation of the x-ray beam is an effective means of shielding both the patient and staff. An elongated cone extending from the tube chassis to the patient's head as described by Latimer and Perrelly[35] is an example of double shielding during cerebral angiography.

Examples of some of the shielding in use in this hospital are shown in figures 1 through 4. Lead aprons are placed over the pelvis in every possible instance, including exposures for radiography of the extremities.

The actual mechanics of patient protection are most often performed by technicians. The physician must constantly supervise and train his technical staff so that repeat examinations are minimized. Instruction of the technical staff should include methods for reducing patient and staff irradiation as well as explaining the significance of protection. Physicians should provide the proper working attitude toward protection and equipment for protective measures, and must insist upon their application in every possible situation.

Selection of Examinations. Selection of the proper examination is an adequate subject for a separate article. We feel that ex-

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tensive "routine" surveys and frequent re-examinations often result from lack of initiative or experience on the part of the requesting physician. This practice is a considerable source

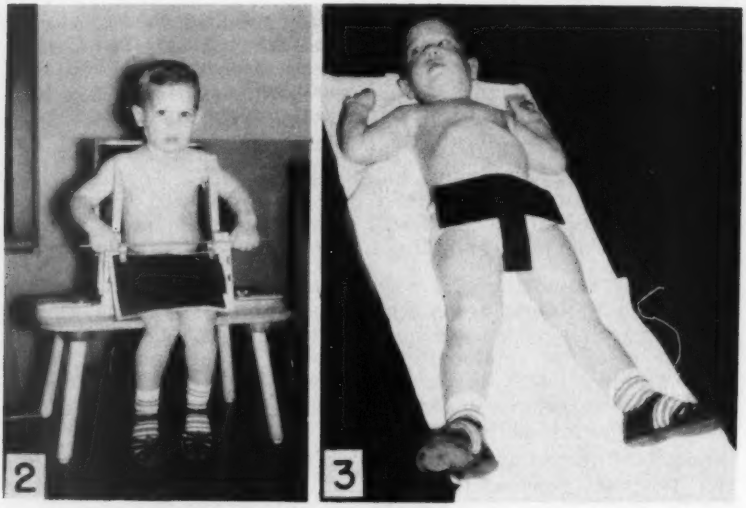

Figure 2. Lead-rubber apron shield added to commercially produced infant chest-film immobilizer. Figure 3. Lead-rubber, T-shaped shield used to protect either the testes or ovaries of children requiring frequent examination of the hip for congenital dislocation.

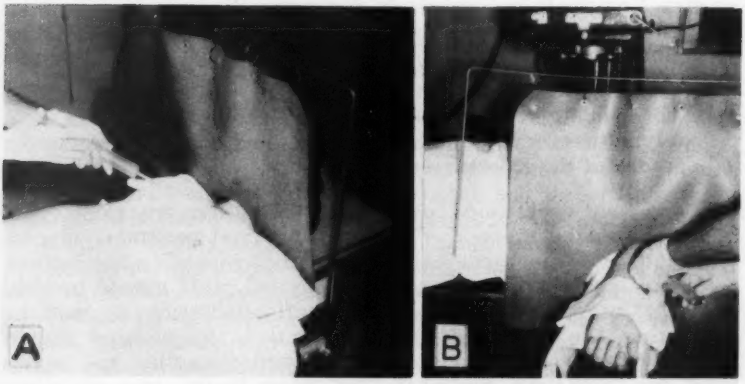

Figure 4. Lead-rubber shield devised to protect both the patient and the physician from scattered radiation during angiography. (A) *Position of shield during cerebral or carotid angiography.* (B) *Position of shield during angiocardiography, retrograde aortography, or cardiac catheterization.*

of radiation exposure. A minimum adequate preliminary survey will often produce the desired information and, if not, additional films can be obtained as may be specifically indicated. Careful evaluation of the information desired and selective examination is a fertile field in which to decrease patient exposure. Radiography should be performed in lieu of fluoroscopy except for the study of cardiopulmonary physiology, gastrointestinal examinations and a few other selected procedures. Based on figures already cited, one minute of chest fluoroscopy gives a radiation exposure equal to 80 routine posteroanterior chest films when both procedures are conducted properly. A similar comparison may be made in the use of Bucky films in lieu of planograms for evaluating a pulmonary cavity.

Patient Selection. Actually, one does not truly select the patient. However, an awareness on the part of the physician of disease incidence, age incidence and the four population groups described earlier will guide him to a degree of selectivity. This expedient for reducing exposure can best be made clear by a few examples. The rapidly healing fracture of a well-set bone in a child requires infrequent radiographs, whereas the older person with slower healing can tolerate frequent x-ray examinations. The young adult with a "stable" duodenal ulcer rarely needs more than an annual gastrointestinal series, but a gastric lesion needs frequent checks. On the other hand, the older adult with a duodenal ulcer should be examined frequently. Who can tell if his latest symptoms are not due to a new gastric lesion?

Multiple films of the abdomen should be avoided in every possible instance during pregnancy.[36] This includes routine pelvimetry. Necessary pelvimetry should be performed as late in the pregnancy as possible. Examination of the abdomen and pelvis during pregnancy should be avoided except under the most severe hazards to the mother's health. Other radiographic procedures, however, can be performed with shielding as needed with minimum or no risk to the fetus. Multiple examinations in the same area, film studies in the occupationally exposed individual, and all x-ray procedures in children should be carefully considered and frugally ordered.

DISCUSSION

The foregoing comments are designed to bring out certain salient points in a common-sense approach to the problem of hazards arising from the medical use of ionizing radiation. (1) It is freely admitted that radiation is harmful as well as helpful. Injuries to the irradiated organism may be derived from either acute or chronic exposure. The same is true of many drugs, sunlight, foods, automobiles, and exercise. (2) The injurious effects of radiation are well documented and rather well understood qualitatively. Our present knowledge does not, however,

permit accurate quantitative evaluation of threshold doses. (3) Certain standards are generally accepted as representing a maximum permissible dose of ionizing radiation per unit of time. Certain maximums have been suggested as relatively "safe" in genetic terms of reference. These are estimates evolved from evaluation and extrapolation of available data and should be considered as working rules rather than scientifically exact criteria. (4) Finally, there are various ways by which physicians can control and reduce radiation exposure to the patient without jeopardizing our current standards of medical practice.

Recent public (and medical) excitation about radiation hazards is largely due to extensive publicity regarding these concepts. This publicity has, in many instances, produced an unreasonable fear of medical radiation in the laity and a rather defensive attitude in some segments of the medical profession.

Obviously, medicine cannot do without radiology. In the first five decades in which x-rays have been used, life expectancy has increased 18.8 years, and undoubtedly medical x-ray has played a part in this extension of life rather than shortening it! Deaths from tuberculosis alone have diminished 184.9 per 100,000 population during this interval. We are justifiably warned that one of the dangers of radiation is fetal death and infant mortality; but stillbirth and infant mortality rates have consistently dropped.[28]

Stone[28] proposes that the dangers involved in the medical use of radiation must be evaluated in relation to the other dangers of modern life. In 1955, motor vehicle accidents killed 38,426 people[37] while March's study of leukemia in radiologists[11] was based on only 14 deaths in 20 years. Yet we do not try to prohibit the use of automobiles.

A practical solution to the problem of hazards in medical radiology would seem to be *education* of the physician who requests or performs radiologic procedures and *education* of the patient population upon which the procedures are performed.

We have shown how the physician can reduce exposure, but responsibility does not end there. He must understand the concepts of medical radiology—its uses, benefits, harmful effects and methods of protection. This understanding need not be an erudite knowledge of radiation physics, but rather should be a general understanding of the problem that will enable the physician to deal intelligently with patients and fellow physicians. It is the physician's task to reassure the frightened patient, order procedures with selectivity, and perform them properly so as to arrive at the true permissible dose: the least amount of radiation to which the patient can be exposed without compromising patient care.

Educational material is readily available for physicians and laymen. Many excellent articles have appeared in recent medical literature. The American College of Radiology has an extensive educational and public relations program on radiation uses and hazards that is available for presentation to both professional and lay groups.

The case for education was ably stated by Dr. Richard H. Chamberlain who said:

> Education of all is a continuing obligation, so that our present knowledge may be extended to its fullest use. To do our work with all of the skill and care that is attainable is the most effective of all measures in radiation protection. This is not to be achieved by legislation or rigid rules, by exhortation, or even fear, but proceeds from the individual mind which employs wisdom, experience, and common sense.[38]

SUMMARY

Ionizing radiation as utilized in medical radiology possesses potentially harmful as well as helpful qualities. The maximum permissible dose of 0.3 r per week or 210 r before age 60 is at best an educated guess and does not include exposure from medical and dental x-rays. The dosage from these sources is considered relatively insignificant to the entire population group when compared to background radiation that cannot be modified. Still, dosage from medical radiation should be reduced to the minimum possible levels consistent with good medical practice. Equipment and procedural methods for achieving this reduction are discussed.

Education of physicians and patients is required to overcome unreasonable fears of medical radiation which, as a result of recent publicity in the lay press, threaten to compromise use of this invaluable medical tool. The hazards of radiation exposure are known, and the means of protection are available.

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MEDICAL PREPAREDNESS IN PEACETIME

Why is it desirable to improve our medical preparedness? Have we not always handled our medical emergencies in both peace and war reasonably well? The answers to these questions are important. If we are already doing all right then let us forget the whole thing. On the war combat side the answer is obvious: things have changed and are changing rapidly. Newer weapons of war can inflict casualties on a scale previously impossible. On the natural disaster side the answer is not so obvious, but it is clear. We have not always done a good job in handling medical emergencies. Advances in medical knowledge in the last quarter-century have frequently not been put to use. It is important that the medical profession realize this and further realize that a peacetime medical emergency can occur any place. We have not learned how to prevent bus, train, or airplane crashes, hurricanes, tornadoes, floods, or fires. If the local medical community will prepare itself to cope adequately with these, it will have gone a long way toward preparing itself for a wartime disaster.—Silas B. Hays: Medical Preparedness. *Journal of the American Medical Association,* September 20, 1958.

EFFICACY OF IMMUNIZATION AGAINST ASIAN INFLUENZA IN THE UNITED STATES NAVY

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BENJAMIN F. GUNDELFINGER, *Commander, MC, USN*

THE far-flung ships of the United States Navy have been a sensitive indicator of the spread of Asian influenza throughout the world. Ships of the Pacific Fleet began reporting epidemics in April 1957, and a high percentage had been involved by August 1957. The A/Japan/305/57 strain of influenza virus was isolated at the 406th Army Medical General Laboratory from the throat washings of a sailor of an aircraft carrier on which an outbreak of influenza occurred following the visit of the ship to Hong Kong in mid-April. Sporadic outbreaks began to occur in the Atlantic Fleet as early as June 1957 but it was not until a large number of ships entered European ports during North Atlantic Treaty Organization (NATO) exercises in September and October that many simultaneous outbreaks were reported. The span of experience thus extends before and after the introduction of specific vaccines containing Asian strains of influenza virus in late August and September of 1957.

These ships have provided the Navy with an opportunity to assess the value of the vaccines made available to them. Ships of the same type are reasonably comparable in number and composition of personnel, intimacy of contact, and daily work loads. Operational assignments provide the major variable because shore contacts are responsible for the introduction of disease aboard ship. Opportunities for shore leave or the rigorousness of operations can greatly influence the willingness of personnel to report illness, and the medical responsibilities for ships in port, particularly smaller ships, are often assumed by larger ships or shore dispensaries. While these factors, together with other variables, prevent the consideration of any one ship as a comparable control for another, the aggregate experience of a number of ships appears to have provided a negative answer to the major question of concern to the Navy, "Would the available vaccines offer significant protection against explosive outbreaks of Asian influenza in operational units of the Navy?"

From the Bureau of Medicine and Surgery, Navy Department, Washington 25, D. C. Capt. Seal is now Commanding Officer, U. S. Naval Medical Research Unit No. 3, c/o American Embassy, Cairo, Egypt.

Comparison of the experience on ships following vaccination with the results obtained in controlled studies among recruits at the Great Lakes U. S. Naval Training Center[1] leads to the inescapable conclusion that the failure of protection on ships resulted only from the poor antigenic potency in humans of certain lots of vaccine obtained commercially.

BACKGROUND

Polyvalent aqueous influenza vaccines have been administered to all naval personnel each fall since 1954. The virus strain compositions of these vaccines have been in accordance with recommendations of the Commission on Influenza of the Armed Forces Epidemiological Board.[2] U. S. Naval Medical Research Unit No. 4 has undertaken controlled studies among volunteers from the recruit population at the U. S. Naval Training Center, Great Lakes, Ill., in an effort to evaluate the effectiveness of these vaccines each winter. Because of the generally low incidence of influenza between 1954 and 1957, efforts at evaluation had not been particularly rewarding, either in these controlled studies or by comparison of military with civilian experience.

In May and June of 1957 extensive outbreaks of Asian influenza occurred on ships operating in the western Pacific. A high proportion of men on these ships had been vaccinated with a polyvalent vaccine six to seven months previously and many had been vaccinated annually for several years. Lacking other control measures, revaccination with the available polyvalent vaccine was advised. This vaccine, which had been used in the annual program during the fall of 1956, contained 250 CCA (chick-cell agglutinating) units each of Swine, FM1 strains of influenza A viruses, and the Great Lakes 1954 strain of influenza B virus per ml. It is hereafter referred to as "1956-polyvalent."

When monovalent Asian influenza vaccines containing 200 CCA units of the A/Japan/305/57 strain per ml became available through commercial channels in late August, a special purchase of 125,000 ml was made. About 90,000 ml were immediately distributed to Navy units scheduled to participate in NATO exercises in the northeastern Atlantic and Mediterranean during September and October. Most of the balance was distributed to other fleet and shore units in Great Britain, Continental Europe, and the Mediterranean areas where influenza was spreading in civil populations, and military populations were under immediate threat. A small quantity was sent to the U. S. Naval Medical Research Unit No. 4 at Great Lakes, Ill., for controlled studies on antigenicity and effectiveness. Thus, monovalent influenza vaccine was available in quantity only to ships and other operational units of the Atlantic Fleet. The dosage directed for personnel of such units was 1 ml subcutaneously. This monovalent vaccine obtained in the standard 5 ml commercial packaging is to be differentiated from two other lots of monovalent vaccine which had been made available to U. S. Naval Medical Research Unit No. 4 for experimental study by two other manufacturers.[1]

Because a polyvalent vaccine had again been planned for use in the fall of 1957 and contracted for in March of 1957, it was necessary to renegotiate the contract and substitute an Asian strain in the formula. The final formula of this aqueous polyvalent vaccine included 200 CCA units each of the Swine, PR301, FM1, Japan/305 strains of influenza A viruses and the Great Lakes 1954 strain of influenza B virus per ml. This vaccine, which became available for use throughout the Navy about 1 October, is hereafter referred to as "1957-polyvalent."

The 1957-polyvalent vaccine was distributed for use mostly at shore installations and in the Pacific Fleet. Operational units were instructed to use a dose of 1 ml subcutaneously, but non-operational personnel and dependents were to receive it intracutaneously. Where used, military personnel received two 0.1 ml intracutaneous injections at different sites simultaneously, dependents only a single 0.1 ml injection.

Data from sources other than the U. S. Naval Medical Research Unit No. 4 cited in the text have been abstracted from the routine message and written special epidemiologic summaries forwarded to the Bureau of Medicine and Surgery, Department of the Navy, by ships and stations upon occurrence of unusual outbreaks of infectious diseases.

OBSERVATIONS

Monovalent 200 CCA Asian Strain Influenza Vaccine

The previously mentioned monovalent Asian strain vaccine consisted of a single lot from one manufacturer. It was distributed in late August to over 200 ships, most of which were at East Coast ports preparing to depart for the NATO exercises. Ninety of these ships made special message reports of outbreaks of Asian influenza at sometime between July 1957 and January 1958. Fifteen outbreaks have been serologically confirmed as to the causative virus. On 41 ships the onset of the outbreak was 10 or more days after the crews were vaccinated, and the cause was confirmed serologically on 9 of them. On 22 ships outbreaks had terminated prior to vaccination of the crews, and three of these were confirmed serologically. In 26 other outbreaks the relationships between the beginning of the outbreak and vaccination date could not be defined exactly or vaccination was begun during or just before the outbreak. Not all of these 90 ships submitted a final written epidemiologic summary giving exact data on personnel strength, total cases, and other details during the period of the outbreak. The attack rates on ships reporting only by message were essentially similar to those submitting both messages and final written reports, but only the latter were utilized in the subsequent analyses.

The fact that 121 ships did not make a special message or written report of influenza was of no significance because it was known that many ships failed to make such reports during

the period in which influenza was widespread. Many of the ships that failed to submit messages could be identified as having experienced epidemics through regular morbidity reports, other ships having named them as contacts, and positive serologic reports from laboratories. Incompleteness of data precluded their further consideration.

In table 1 are summarized data on the attack rates during outbreaks of influenza which had subsided prior to or which began 10 or more days after vaccination. Summaries from all ships included claimed that all personnel, with the exception of an occasional individual sensitive to eggs, were vaccinated with 1 ml of the monovalent vaccine subcutaneously.

TABLE 1. *Asian influenza on U. S. Navy ships*

Class of ship	Number of ships reporting	Total strength	Total cases	Attack rate			
				Epidemics prior to vaccination*		Epidemics after vaccination*	
				Average	Range	Average	Range
Battleships	1	2,340	748	31.9	-		
Aircraft carriers	1	3,495	631	18.1	-		
Cruisers	1	1,505	355	23.6	-		
Destroyers	6	1,465	653	44.5	15.3 to 60.6		
Other	4	2,644	1,014	38.3	15.3 to 48.0		
Total	13	11,449	3,401	29.7	15.3 to 60.6		
Battleships	1	1,534	489			31.7	-
Aircraft carriers	3	8,624	2,462			28.6	22.6 to 30.9
Cruisers	2	3,105	829			26.6	22.6 to 29.9
Destroyers	19**	4,448	1,651			37.3	12.2 to 62.3
Other	9	3,324	1,062			31.9	8.7 to 64.7
Total	34	21,035	6,493			30.7	8.7 to 64.7

*Only ships submitting final summaries on epidemics terminating prior to or beginning 10 or more days after subcutaneous inoculation of crews with a 200 CCA/ml monovalent Asian influenza vaccine included.
**Excludes two that reported two separate epidemics.

From this tabulation it seems clear that while there was considerable variation in attack rates between ships this was unrelated to whether or not the crews had received the monovalent Asian strain influenza vaccine. The attack rates in the aggregate personnel were remarkably similar despite this variation in individual units. It was also evident from the reports that no major modification of illness occurred, as judged by the range of fever, duration of illness, or variety of symptoms, in those who developed influenza before and after a vaccination program. Attack rates and clinical descriptions of the disease reported from ships where vaccination was begun during epidemics or at some unspecified time in relation to it were consistent with the experience of either group of ships in table 1.

A few specific instances are cited as examples of these shipboard outbreaks occurring 10 or more days after vaccination with the monovalent influenza vaccine. The entire crew of a submarine received 1 ml of the vaccine subcutaneously on 3 October. An outbreak began on 20 October, a few days after tying up along-

side a sister submarine on which influenza was occurring. Sixty per cent of the crew became ill in the next 10 days, many with fever to 103° to 105°F. The outbreak on the sister submarine began on 14 October and involved 65 per cent of her crew; influenza vaccine was not administered on this submarine until 17 October. The crew of a fast attack transport were given 1 ml of the monovalent vaccine on 27 August and 1 ml of the 1956-polyvalent vaccine on 30 September. An outbreak involving 62 per cent of her crew began on 6 October while at Taranto, Italy. Four of five pairs of sera submitted from this ship to a Navy Preventive Medicine Unit exhibited diagnostic Asian strain antibody titer rises. A final example is that of an aircraft carrier whose crew was vaccinated subcutaneously with the monovalent vaccine on 3 September. During the next 11 days the ship was at sea. On 20 September, six days after arrival in Scotland, an influenza outbreak began in which 31 per cent of her crew were involved by 15 October. Diagnostic increases in Asian strain antibodies occurred in five of six pairs of sera submitted to a Navy Preventive Medicine Unit.

The first or acute sera of previously vaccinated patients from these ships which were submitted to laboratories for diagnostic studies were devoid of antibodies against the Asian strain at the lowest dilutions used in the tests. This coincided with the results of antigenicity studies carried out at Great Lakes (table 2). Neither did controlled studies at Great Lakes using this commercial monovalent vaccine exhibit any evidence of significant protection.

Reports of influenza outbreaks occurring between April and August 1957 were received from 16 other ships, including destroyers, a cruiser, and aircraft carriers. All of these ships were operating in the Pacific at the time of these outbreaks except for two destroyers which were at Newport, R. I. The attack rates reported ranged from 13.8 to 66.3 per cent on individual ships with an average of about 30 per cent.

These data indicate that the experience of those ships which received monovalent vaccine was consistent with that of ships which became involved before the vaccine was available. The vagaries of spread of the disease among personnel of ships which accounts for the variation in attack rates cannot be explained other than to state that the variation appears to be real and not entirely due to differences in diagnostic criteria or reporting. Several ships have reported more than one outbreak, and one (to be described later in this article) had two separate confirmed outbreaks. Such variation, however, points to the difficulties in evaluation of prophylactic measures within a small group of ships, and offsets many of the individual opinions which were expressed pro or con by individual commands about the effectiveness of the vaccine on the basis of comparison with other ships in the vicinity.

TABLE 2. *Hemagglutination-inhibition antibody titer response to influenza vaccines in naval personnel, October to December 1957*

Vaccine	Dose and route	Number of subjects	A/Japan/305/57			A/Great Lakes/1148/53			B/Great Lakes/176054		
			Per cent with titers		Geometric mean	Per cent with titers		Geometric mean	Per cent with titers		Geometric mean
			≥10	≥20		≥10	≥20		≥10	≥20	
1956-polyvalent	1.0 ml subcutaneously	12	(0)*	(0)	(<10)	(50)	(25)	(10)	(100)	(83)	(21.5)
			8	0	<10	67	67	33.5	100	100	100.0
1957-polyvalent	1.0 ml subcutaneously	65	(11)	(3)	(<10)	(39)	(29)	(12.4)	(55)	(50)	(28.5)
			28	20	<10	49	33	16.3	57	53	35.0
1957-monovalent	1.0 ml subcutaneously	35	(3)	(0)	(<10)	-	-	-	-	-	-
			9	6	<10						
1957-polyvalent	0.2 ml intracutaneously	42	(24)	(7)	(<10)	(21)	(10)	(<10)	(38)	(38)	(36.3)
			43	31	10	31	25	16.5	40	38	51.0
1957-monovalent	0.2 ml intracutaneously	37	(0)	(0)	(<10)	-	-	-	-	-	-
			5	3	<10						

* Parenthesis = prevaccination.
No parenthesis = postvaccination.
(14 to 30 days)

Shipboard Experience With the 1957-Polyvalent Vaccine

Due to the late distribution of this vaccine and the fact that most ships in the Atlantic Fleet received the monovalent vaccine, relatively few instances were reported where it could be ascertained that this vaccine had been given 10 or more days prior to beginning of an outbreak.

Four destroyer type ships submitted final epidemiologic summaries indicating outbreaks had begun 10 to 30 days after the crews were vaccinated with 1 ml subcutaneously. There were 256 cases among the total of 793 men on these four ships. The total attack rate was 32.3 per cent with variation on individual ships from 15.3 to 54.2 per cent.

An attack transport reported an epidemic during a cruise to Alaska in October in company with a sister transport. The attack rate on the reporting transport, personnel of which were unvaccinated, was 36.2 per cent. On the sister transport, personnel of which had been vaccinated 7 to 10 days prior to beginning of the outbreak, the attack rate was 32.6 per cent. The report concluded: "On the basis of this limited study, there appears to be no significant decline in incidence between inoculated protected (ship)_____ and control (ship)_____."

A destroyer escort reported immunization of its crew of 151 men on 25 September, with 1 ml of 1957-polyvalent vaccine subcutaneously. Epidemic influenza began on 24 October. A total of 81 cases occurred, an attack rate of 53.6 per cent. This outbreak was not confirmed as to cause but occurred while the ship was alongside another in which a confirmed outbreak was occurring. Another destroyer escort reported an attack rate of 54.2 per cent in the crew which had been vaccinated subcutaneously 10 days prior to the beginning of an outbreak. In contrast, two destroyers with outbreaks beginning 14 or more days postvaccination reported attack rates of 15.3 and 20.6 per cent and a cruiser reported only 9.8 per cent of its crew involved. There were no notable differences in the clinical description of disease occurring aboard these ships from those of unvaccinated ships and the only conclusion that can be reached is that the disease did occur in high incidence and without major modification of severity among personnel vaccinated 10 or more days before the beginning of outbreaks.

Interpretation of additional reports was complicated by the possibility that ships were undergoing a second outbreak of influenza rather than initial outbreaks during the postvaccination period. U. S. Naval Medical Research Unit No. 2[3] investigated an outbreak occurring in a destroyer division in the Philippines during November and December. Men on the four destroyers involved had received 1957-polyvalent vaccine between 17 and 22 October and the 1956-polyvalent vaccine in August of 1957. Each of the ships had experienced an outbreak of influenza-like illness during June and July of 1957 while operating in the

Long Beach, Calif. area. In November, one destroyer had an attack rate of 49.5 per cent despite the possibility that some personnel previously had had the disease and that both 1956- and 1957-polyvalent vaccine had been administered in the three-month period prior to the beginning of the second outbreak. The destroyer on which the fewest cases had occurred at the time of the investigation had experienced the highest attack rate (approximately 15 per cent) during the July outbreaks.

Of interest in this regard was the report from an aircraft carrier in the Pacific. Between 1 and 18 July, while operating in the San Diego, Calif. area, an outbreak of influenza-like illness occurred with 227 cases, or an attack rate of 10.4 per cent. Of the 227 cases, 157 (69 per cent) were admitted to sick bay. The distribution of maximum oral temperatures in these 157 cases is shown in table 3. The average duration of illness was 3 days.

TABLE 3. *Distribution of maximum oral temperatures in hospitalized patients during two confirmed outbreaks of Asian influenza on same aircraft carrier*

Maximum oral (°F) temperature	Patients with elevated temperatures			
	First outbreak (July)		Second outbreak (November)	
	Number	Per cent	Number	Per cent
104	14	8.9	20	11.0
103	38	24.2	43	23.7
102	63	40.1	78	43.1
101	24	15.3	27	14.9
100	12	7.6	11	6.1
99	4	2.6	1	0.6
98	2	1.3	1	0.6
Total	157	100.0	181	100.0

All men admitted had received the 1956-polyvalent vaccine during the preceding winter. A second epidemic occurred on this carrier between 1 and 27 November while it was operating around Japan with a total of 447 cases, or an attack rate of 16 per cent. Of the 447 cases, 181 (41 per cent) were admitted and the distribution of oral temperatures in these 181 men is also shown in table 3. The average duration of illness was 4 days.

The 1956-polyvalent vaccine had been administered to the crew of this carrier between 20 and 25 August. The 1957-polyvalent vaccine was given on 14 and 15 October. The latter vaccine was given intracutaneously, each man receiving 0.1 ml in the volar surface of each forearm, a total of 0.2 ml. On the basis of the higher attack rate and clinical similarity of patients ad-

mitted during the two epidemics, medical officers were of the opinion that the two doses of polyvalent vaccine, one of which contained 200 CCA units of Asian influenza virus, had been ineffective in preventing or ameliorating the disease. No comment was made in the epidemiologic reports from the ship as to the lesser percentage of total cases admitted during the second outbreak. This could have been a reflection of milder disease, or simply exhaustion of hospitalization facilities due to the greater number of cases. Three pairs of sera collected from patients ill in July were reported by the Sixth Army Medical Laboratory as showing diagnostic rises in antibody titer for the Asian strain. Asian strains of influenza were isolated from 9 of 11 throat washings collected by a team from U. S. Naval Medical Research Unit No. 2 during an epidemiologic study of the November outbreak. Diagnostic antibody rises occurred in 30 of 36 pairs of sera.[3] Only one man, of those with acute tonsillitis during the November outbreak, had been ill in July. These observations suggest that illness during the July outbreak conferred relatively complete protection in November.

Results of Controlled Studies on Effectiveness of Vaccine at the U. S. Naval Training Center, Great Lakes

Figure 1 shows the reported febrile respiratory disease rates in naval recruits at Great Lakes two or more weeks after subcutaneous and intracutaneous inoculation with either a placebo solution or the commercial monovalent Asian influenza vaccine. During the period of these studies, Asian influenza was diagnosed in 17 to 89 per cent of hospitalized cases tested by serologic methods or virus isolation technics. The intracutaneously inoculated group experienced an average reported illness rate of 63.4 per 1,000 per week during the study period, as compared to 84.3 per 1,000 per week in its placebo control group. The illness rate in the subcutaneously vaccinated group was 54.9 per 1,000 per week as compared to 59.4 per 1,000 per week for its control. The maximum illness rates occurred somewhat later in the vaccinated men than in the control groups, which suggests that the use of this vaccine may have modified the behavior of influenza in these groups. Undoubtedly, the slight degree of protection observed would be detected only under controlled conditions.

In contrast to the poor results observed with the monovalent vaccine distributed to naval activities in the Atlantic and Mediterranean areas are those obtained using a monovalent Asian strain vaccine prepared by another manufacturer. This latter vaccine contained 200 CCA units of A/Japan/305/57 strain per ml and was administered subcutaneously in a 1 ml dose. Summary results of this study, the details of which were reported elsewhere,[1] are shown in figure 2. The reported febrile respiratory disease rates in naval recruits inoculated with either the experimental Asian vaccine, the 1956-polyvalent vaccine, or a control

solution, are shown for each week of the study period. It is apparent that a marked reduction in reported respiratory illness was associated with the use of this monovalent vaccine. The 1956-polyvalent vaccine failed to prevent an outbreak of respi-

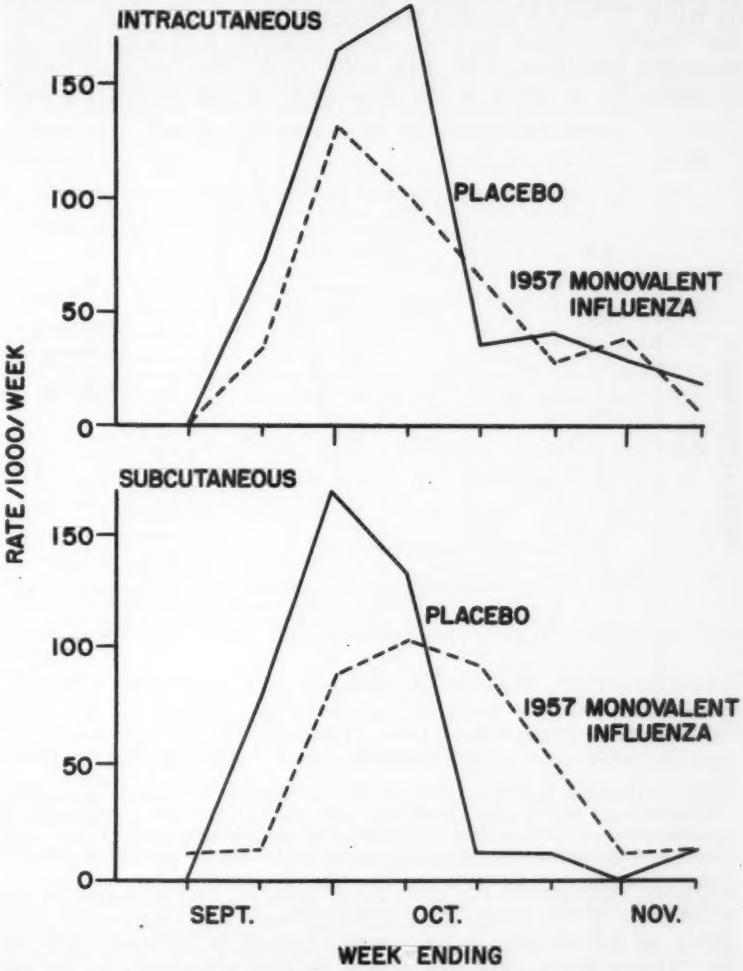

Figure 1. Reported febrile respiratory disease rates in naval recruits two or more weeks after subcutaneous or intracutaneous vaccination with 1957-monovalent Asian vaccine or placebo solution.

ratory disease in the population in which it was used; however, the slight, but statistically significant, reduction in illness rates in this group is of considerable interest in connection with the immunology of influenza.

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Results of Human Antigenicity Studies with Several Vaccines Used in the Navy

The antibody response to vaccination with the vaccines used at Great Lakes and with the commercial vaccines was examined by the U. S. Naval Medical Research Unit No. 4. Table 4 shows the results of these tests in naval recruits. It appears that the protamine precipitated vaccine (vaccine A) was the most potent antigenically in terms of the percentage of men responding with

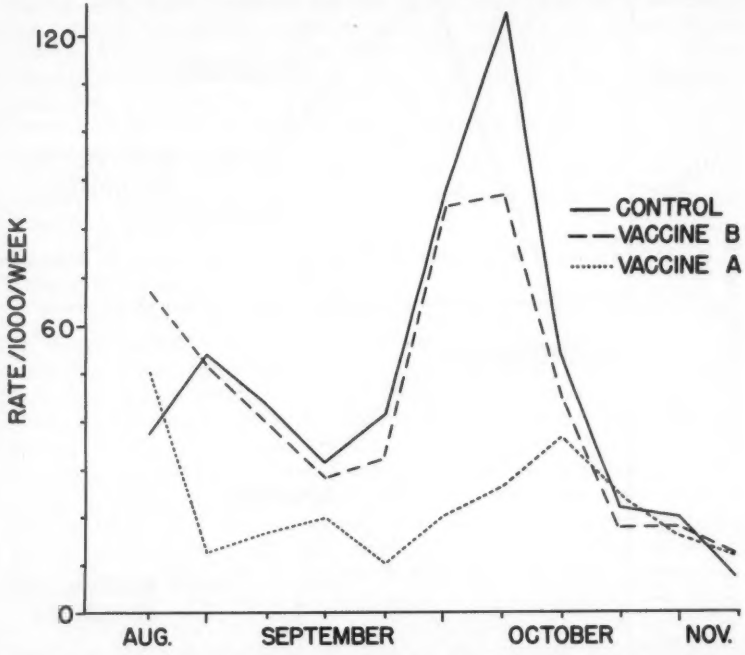

Figure 2. Reported febrile respiratory disease rates in vaccinated and control naval recruits two or more weeks after vaccination. Vaccine A = monovalent Asian vaccine; vaccine B = 1956-polyvalent influenza vaccine. (Reproduced with permission from New England Journal of Medicine, *November 20, 1958.*[1])

antibody titer levels of 10 to 20 or greater, and in terms of the mean titer of the group after subcutaneous vaccination, as compared to the results in its placebo group. Of interest here are the results using vaccines B and C. Forty-five per cent of the men receiving vaccine B had antibody titers of 20 or greater two weeks after subcutaneous vaccination, while none in their placebo counterpart had antibody titers of this magnitude. The data indicate that vaccine C, the vaccine issued to naval units in the Atlantic and Mediterranean areas, was a comparatively poor antigen when given either subcutaneously or intracutaneously. The effectiveness of vaccines B and C in the prevention

of acute respiratory disease was investigated in field trials at Great Lakes. As stated above, only slight clinical protection was observed in association with the use of vaccine C. Vaccine B, on the other hand, was highly efficacious in reducing acute febrile respiratory illness rates. Thus, prophylactic efficacy of these vaccines correlated well with their antigenic potency with respect to the epidemic viral strain. Unfortunately, no observations on the efficacy of vaccine A are available because the population in which it was being studied was no longer under observation during the period of influenza prevalence at Great Lakes.

TABLE 4. *Antibody response to monovalent Asian strain influenza vaccine in naval recruits 14 to 21 days after vaccination, Great Lakes, Ill., July to September, 1957*

Vaccine	Dose and route	Vaccinated				Control			
		Number of subjects	Per cent with titers*		Geometric mean titer	Number of subjects	Per cent with titers		Geometric mean titer
			≥10	≥20			≥10	≥20	
A	1.0 ml subcutaneously	19	84	79	40.0	10	20	10	<10
B	1.0 ml subcutaneously	33	61	45	14.2	31	3	0	<10
C	1.0 ml subcutaneously	27	33	14	<10				
	0.1 ml intracutaneously	50	18	8	<10	21	5	5	<10
	0.2 ml intracutaneously	25	48	20	<10				

*Reciprocal hemagglutination-inhibition antibody titers to A/Japan/305/57.
Vaccine A = Protamine precipitated vaccine, 500 CCA units per ml.
Vaccine B = Aqueous vaccine, 200 CCA units per ml.
Vaccine C = Aqueous vaccine, 200 CCA units per ml. Vaccine used by naval units in Atlantic and Mediterranean areas.

Serum specimens were obtained by several Navy Preventive Medicine Units from men stationed at shore stations and aboard ships in the Pacific, Atlantic, and Continental United States areas, before and after inoculation with influenza vaccines procured from commercial sources by the Navy. These specimens were tested for influenza hemagglutination-inhibition antibodies by U. S. Naval Medical Research Unit No. 4. Table 2 shows the results of these tests. The antibody titer response to the 1956-polyvalent vaccine, which did not contain Asian influenza antigen, was significant to an A-prime and a B-influenza antigen. As might be expected, no titer response to an Asian strain antigen was observed with this vaccine. The antibody titer response to the 1957-polyvalent and monovalent vaccines was uniformly poor, even to A-prime and B-influenza antigens also contained in the polyvalent vaccine, and with which these men presumably had had prior experience. The data suggest, however, that the 1957-polyvalent vaccine may have produced a better Asian strain antibody response than did the monovalent vaccine. These results suggest that the vaccines in use by the Navy in the fall of 1957 were relatively impotent antigenically. The results of the con-

trolled study with one lot of these vaccines at Great Lakes support this conclusion.

DISCUSSION

Several reports have appeared which establish beyond question that a 1 ml dose of aqueous vaccine containing about 200 CCA units of an Asian strain of influenza virus will reduce respiratory illness morbidity during epidemics of Asian influenza.[1-2, 4-5] With one exception, these reports have been preliminary and based on the incidence of gross acute respiratory illness. Variation in apparent degree of protection between the several studies is as yet unassessable because this will depend on the Asian influenza specific attack rates in the experimental populations. In the one study in which such rates have been measured, human volunteers were experimentally infected and the comparability of results to what might be expected in naturally occurring disease is unknown.[4]

The controlled studies by U. S. Naval Medical Research Unit No. 4, among recruits at Great Lakes using a commercially prepared vaccine have also shown reduction in total acute respiratory illness during an epidemic of Asian influenza.[1] In contrast, the somewhat limited trial of monovalent vaccine prepared by another manufacturer obtained through normal military procurement channels provided no such reduction. From the data presented herein, it seems clear that this same monovalent vaccine was virtually worthless from the standpoint of any significant degree of protection or modification of illness when used in the personnel of ships. The same assessment would also seem to apply to the 1957-polyvalent vaccine containing the Asian strain which was a product of the same manufacturer. While there was some suggestion that reduction in the incidence of illness followed use of the 1956-polyvalent vaccine, it was at most very minor and of no practical significance.

The Commission on Influenza of the Armed Forces Epidemiological Board has described the difficulties encountered by the manufacturers in producing a vaccine of the desired potency, and in the assay of potency by the usual methods. They have also interpreted the results of controlled studies on antigenicity and effectiveness of the early products as indicating that vaccines assayed as containing 200 CCA units of Asian strain influenza viruses were "suboptimal."[2] In view of this basic inadequacy of the vaccine and the technical difficulties surrounding its manufacture and potency testing, it is not surprising that one or more lots of vaccine should be of such low potency as to be essentially valueless. The open question is to what extent this occurred, and how many other groups shared the Navy experience with commercially released lots of vaccine? Even if it should be ascertained that the Navy experience was unique, there would seem to be a clear indication that more research on methods of production and assay is needed, and that field testing should include vaccines procured for general use. Normal

governmental procurement procedures require competitive bidding and acceptance of the lowest bid(s). This in itself will require the successful bidder to shave production costs to a minimum. At present, there is grave doubt as to the abilities of either the producer or receiver to assay the probable protective value of the vaccine with any high degree of accuracy, on the basis of a chicken erythrocyte agglutination test.

There seems to be a direct relationship between the ability of vaccines to stimulate antibodies against the Asian strain in humans and the protective efficacy of the vaccine. In human volunteer studies, Bell and co-workers[4] found some degree of correlation between the antibody titers and the development of febrile illnesses following inoculation with Asian influenza viruses. The relatively good antibody response in Navy recruits from vaccines providing protection is sharply contrasted to the weak responses following vaccines that provided little, if any, protection. Antibody rises following infection have generally exceeded by a considerable degree those following any of the 200 CCA unit vaccines, and there appears to be relatively solid immunity for at least several months following the natural infection. Vaccination with the older polyvalent vaccines did not stimulate measurable antibody against the Asian strain. While there might have been a little protection conferred by this vaccine, if the experience among recruits is not to be written off as a chance variation, it was so slight as to have no more than academic significance.

The failure to obtain definitive information on the comparative protection afforded by intracutaneous and subcutaneous administration of the vaccines was disappointing. The poor antigenicity of the available vaccines, and failure of protection with the subcutaneous route, would have precluded comparisons, even had it been possible to obtain and distribute vaccine at an earlier date. Because of the importance of this question, when decisions must be made in utilization of inadequate supplies of vaccines to meet requirements by the usual dosage, more information is necessary. It was surprising to encounter such conflict in opinions and so little definitive data at the time decisions had to be made in the early fall of 1957.

This partial summary of the Navy's experience with Asian influenza and specific vaccines against these strains of influenza viruses has emphasized both the military need for an effective vaccine and the inability of the vaccines procured through normal procurement channels in the early fall of 1957 to meet this need. Undoubtedly, the later vaccines would have provided a higher degree of protection but the delay in their availability was far too long for military purposes. When the next new variant appears, if critical operational units vital to the military defense of the country are still scattered throughout the world, some quicker way must be found to provide effective protection, if defense is not again to be jeopardized. These units are involved

as the disease passes through the country in which they are located, and there is little time available between first appearance of the variant and the involvement of critical military populations.

SUMMARY

Epidemics of Asian influenza occurred on Navy ships and stations as the disease spread from the Far East around the world. This afforded records of outbreaks in relatively comparable populations before and after the initiation of vaccine programs in the early fall of 1957. In addition, controlled studies with several vaccines were conducted among recruit volunteers at the U. S. Naval Training Center, Great Lakes, Ill.

Monovalent vaccines containing 200 CCA units of Asian strain influenza virus per ml, prepared by two different manufacturers, were tested by U. S. Naval Medical Research Unit No. 4 at Great Lakes. One of these was highly effective in reducing attack rates for febrile respiratory illness during an epidemic of Asian influenza, the other was not effective. The same monovalent vaccine from the third manufacturer did not appear to provide any significant protection from, or modification of, illness caused by Asian influenza viruses when used routinely in personnel of ships of the Atlantic Fleet.

Reimmunization with older polyvalent vaccines not containing Asian influenza virus, which had been used routinely during the winter of 1956-1957, did not provide any practical reduction in attack rates for Asian influenza. Major outbreaks also occurred in personnel who had been immunized by either the subcutaneous or intracutaneous routes with a new polyvalent vaccine containing 200 CCA units of Asian strain of influenza virus. The ability of vaccines to stimulate significant increases in hemagglutination-inhibiting antibodies in man was correlated with their protective ability. Some evidence was obtained which indicated that a relatively solid immunity persists for at least four months following illness with Asian influenza.

The experience has emphasized the need for further research on methods for production and assay of vaccines containing new variants of influenza viruses, if adequate protection of military forces throughout the world is to be envisaged when the next major variant appears.

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THE NEED FOR MEDICAL STATESMANSHIP

Do we still believe in better standards of medical education and medicine, or are our ideals changing and does a commercial economy occupy an increasingly prominent place in medical ethics? Are we protecting mediocrity? . . . Nor is it medicine only that faces this issue today but also the dental branch of the healing arts. We are at a parting of the ways—one points toward true statesmanship; the second toward defense of the weaker elements and continued defense of procedures and methods that worked well 30 and 40 years ago but now need readjustments, or revitalization if you will, along with the changing picture of our general economy. In my opinion this second road, the vigorous defense of the "status quo ante," encourages the development of socialized medicine and dentistry in spite of its protagonists who themselves may fear the role of true statesmanship in our profession. Quite the contrary, if we wish to prevent such socialization, then we must all face the question squarely, exert leadership, and promote cooperation between our profession and local, State and Federal governments. If there is continued resistance with hesitancy to lead and develop plans, we shall surely be overwhelmed.—Frank B. Berry: Problems Confronting Medicine Today, *Virginia Medical Monthly*, October 1958.

TREATMENT OF ACUTELY COMPRESSED VERTEBRAL BODY BY IMMEDIATE PROGRESSIVE MOBILIZATION

ERNST DEHNE, *Colonel, MC, USA*
JAMES J. SCHUBERT, *Captain, USAF (MC)*

COMPRESSION of the thoracic and lumbar vertebrae is the result of sudden flexion from acute trauma or stress. It is a common injury, especially in the aged female. This injury in the aged usually is caused by minimal trauma or stress, the osteoporotic bone being susceptible to damage. In the young, however, the fracture usually is associated with more severe trauma, especially when resulting from automobile accidents or falls from significant heights. All patients complain of local vertebral tenderness and generalized paraspinal myalgia. There may be a transitory abdominal distention or urinary retention.

Since the advent of radiographic control in fracture reduction, there has been rather uniform agreement in the management of these injuries. Precise reduction and prolonged immobilization in hyperextension until consolidation in anatomic position occurred were insisted on by Davis,[1] Böhler,[2] Watson-Jones,[3] and Rogers.[4] Minor variations of treatment have been advocated, but generally they all fall into the same method.

This article is not addressed to those who are following the teachings of these masters, who are anatomically reducing the fractured vertebrae and maintaining the reduction by continuous immobilization until the fracture has consolidated in anatomic position. We do not wish to take any position in regard to the necessity of precise reduction or the feasibility of maintaining it, although the following paragraphs will indicate that patients do quite well without reduction.

This article does deal with the relative merits of the prevalent method of treatment of the fractured spine which seems to have evolved in practice without particularly following the principles established by the above-mentioned authors; which form of treatment consists of the application of a body cast, using one of the commercial devices designed for the application of a

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"hyperextension cast." Roentgenograms taken through these casts reveal that the original position resulting from the injury had not been changed substantially. These casts are then maintained for a variable period of time, which usually falls considerably short of the period of immobilization necessary to guarantee consolidation after reduction. The body cast is then followed by some form of brace to be worn for a period of time which the physician considers adequate. The over-all results of that management are satisfactory in a large percentage of cases so treated.

A residual group of patients, however, remain with a variable degree of complaints which fall into what may be termed the sociosomatic sphere. Somatic findings are evident on roentgenographic examination and reveal consolidation of the vertebrae in exactly the same position as at the time of the injury, and which had been maintained in a body cast after injury, plus variable findings such as degenerative changes in one or both the adjoining disk spaces and frequently spontaneous attempt toward body fusion by calcification of the longitudinal ligaments. Functional results, however, vary widely with the patient's character and behavior patterns. Those intent to conquer the world as athletes or others ambitious to lead in their professional fields will have no residual complaints. Persons who are insecure or lacking in ability to cope with the challenge of life may revert to leaning upon society for partial or total support. The complaints are real enough to the individual for unless they, themselves, are convinced of the justification of their claims, they could not in all honesty expect society to offer its breast. There is strong evidence suggesting that the incidence of sociosomatic complaints rises in direct proportion to the duration of the management of the underlying fracture. The current study pertains to the feasibility of shortening the period of treatment of the fracture, thus reducing time of exposure and incidence of sociosomatic problems.

In recent years, Nicoll,[5] in England, has presented a large series of patients in which no attempt was made at reduction and no immobilization was used. His results compared favorably with those of hyperextension and immobilization, and had several obvious advantages: Not only did the patient have a more comfortable method of treatment but was returned to full activity and employment much sooner. In addition, the sociosomatic neuroses of the painful and weak back, with resultant compensation and insurance claims, were greatly reduced.

Also, in the literature, many references are found to the fact that even after reduction with hyperextension and prolonged immobilization in plaster, the majority of vertebral bodies appear on roentgenographic examination essentially as they did at the time of the original injury.[6] Hence, it would seem that the advantage of simple bed rest should be more thoroughly investigated.

MATERIAL

During the period 1953-1957, a total of 35 patients were hospitalized because of isolated acute compression fractures of the thoracic and lumbar vertebrae. The patients fell into two major treatment groups. One group consisted of 12 patients whose initial treatment was begun elsewhere and by hyperextension body casts. These patients were seen at Letterman Army Hospital for follow-up care only. The second group was comprised of 23 patients who were treated primarily at this hospital. All patients in this second group were treated with immediate progressive mobilization. Patients with associated fractures of the long bones are not included unless the associated fractures or injuries did not affect our method of treatment. Likewise, patients on ancillary services who were seen on consultation were not included, due to lack of control. Our series did not include those with compression fractures resulting from osteoporosis or electroshock therapy.

METHOD

The concept of immediate mobilization in major injuries of the extremities has long been advocated and practiced by the senior author. The relief of pain in the injured extremity by immediate motion is thought to be due to a specific action of active motion in decreasing inflammatory reaction.[7] During the period of 1953-1957, this concept was applied to all patients suffering acute compression fractures of the thoracic and lumbar spine who were treated under the direct supervision of the orthopedic service. In this rapid treatment program we are guided by two observations. First, that fractures not only affect the immediate weight-bearing function of the injured bone but simultaneously produce a physiologic interference with the muscle function of the affected area. Initial measures, therefore, were aimed at immediate rehabilitation of muscle function, which is carried out with the patient at strict bed rest and by practicing rolling over from one side to another. This usually can be accomplished within the first two days, and successful return of muscle function is announced by disappearance of pain. Second, pain is considered a part of an over-all inflammatory reaction and is respected in the same sense as the flashing red light at the railroad crossing. The recurrence of pain after normal muscle function has returned indicates that the fracture has been subjected to undue stress. Future progress of the patient, therefore, is regulated on the basis of permitting any function which can be carried out without eliciting pain. It is quite revealing that ambulation, at first in limited periods and distances, can thus be resumed within a few days after injury. We have found, however, that tolerance for prolonged sitting or standing is much slower to return and should be restricted during the early weeks of convalescence. Following the injury, the patient is told that he has to regulate carefully his activities in that respect. We

Figure 1. (A) *Appearance of first lumbar vertebra at time of injury.* (B) *Roentgenogram made through plaster hyperextension jacket 40 days after injury.* (C) *Roentgenogram made 90 days after injury. Back brace was used for next four months.* (D) *Six months after injury. Total treatment, 90 days in body cast, 120 days in brace.*

have found that ambulation and exercise can be carried out with impunity if these criteria are kept in mind.

RESULTS

Results are tabulated in table 1. The average number of days at absolute bed rest was 10, while total days of hospitalization averaged 18. This time frequently included administrative processing following completion of treatment. In civilian practice,

TABLE 1. *Duration of treatment of patients with compressed vertebral fractures*

Treatment	Number of patients	Average age (years)	Average time (days)			Total average treatment time (days)
			In cast	In brace	At bed rest	
Hyperextension jackets	12	26	57	120	-	177
Immediate mobilization	23	41	-	-	10	18

total hospitalization would more closely approximate bed rest time. In the 12 patients seen who had been treated with hyperextension body cast, the average time in plaster was 57 days.

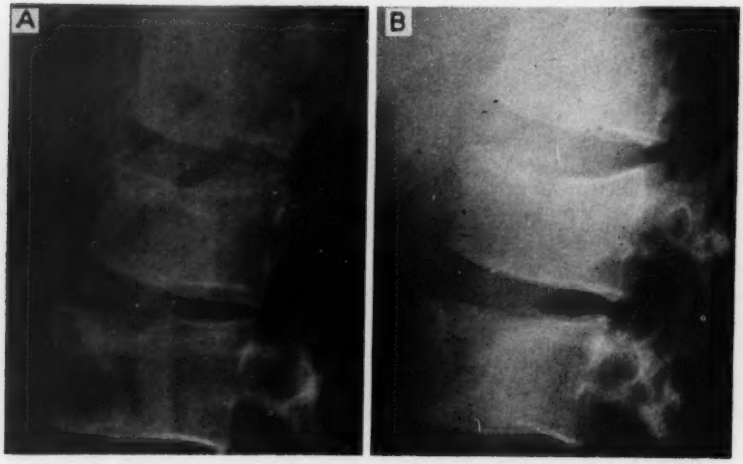

Figure 2. Roentgenograms showing (A) *appearance of vertebra at time of injury and* (B) *60 days later. Patient wore body cast for 60 days.*

In addition, 10 more days were required on the average to properly mobilize the patient and rehabilitate him sufficiently to return to duty. Patients treated with progressive immediate mobilization returned to duty on the average of 18 days following injury. All were free of pain with a normal range of motion. A

restricted profile preventing any undue stress to the low back was given patients for varying periods of time, usually from three to six months.

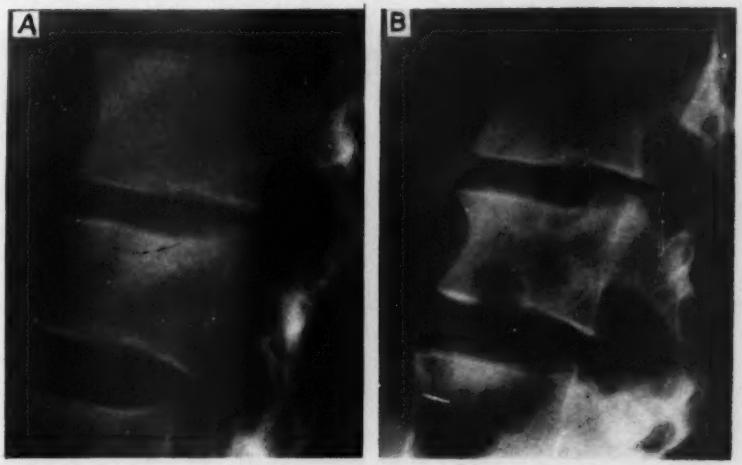

Figure 3. Roentgenograms (A) *at time of injury (treatment by immediate mobilization) and* (B) *60 days after injury when patient was asymptomatic.*

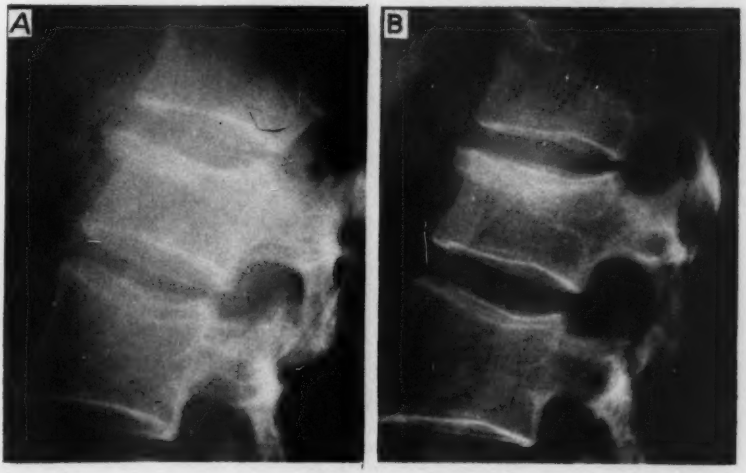

Figure 4. Roentgenograms (A) *at time of injury and* (B) *90 days after injury when patient was asymptomatic. The total time spent in bed was 14 days.*

In many instances follow-up in our series was not possible because of changes in military assignments. However, some were followed for periods up to two years. Comparison of the appearance of the wedged vertebrae with both methods was

possible. Figures 1 through 6 are representative of each. It is apparent that reduction accomplished by hyperextension frequently is lost with the immobilization technic. In most cases,

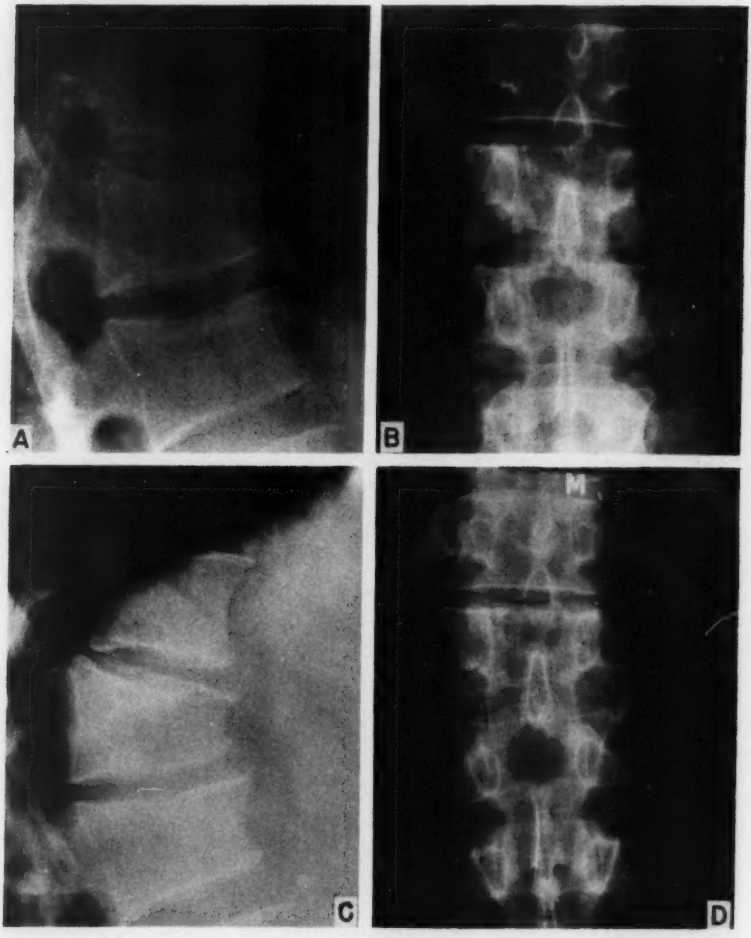

Figure 5. (A) *and* (B) *Appearance at time of injury with treatment by immediate mobilization.* (C) *and* (D) *Two years following injury. Normal function retained.*

reduction was not made at all but the patient was continued in a hyperextension cast. In those cases treated by immediate progressive mobilization, no great change in appearance of the fracture was noted. Anterior compression was not increased.

COMPLICATIONS

Each case was studied for untoward complications. No patient treated by immediate mobilization developed neuropathy during treatment. One patient developed a cord bladder six months following his injury and was subsequently found at laminectomy to have an arachnoid scar. It was believed that this probably was not related to treatment, but due to the original trauma.

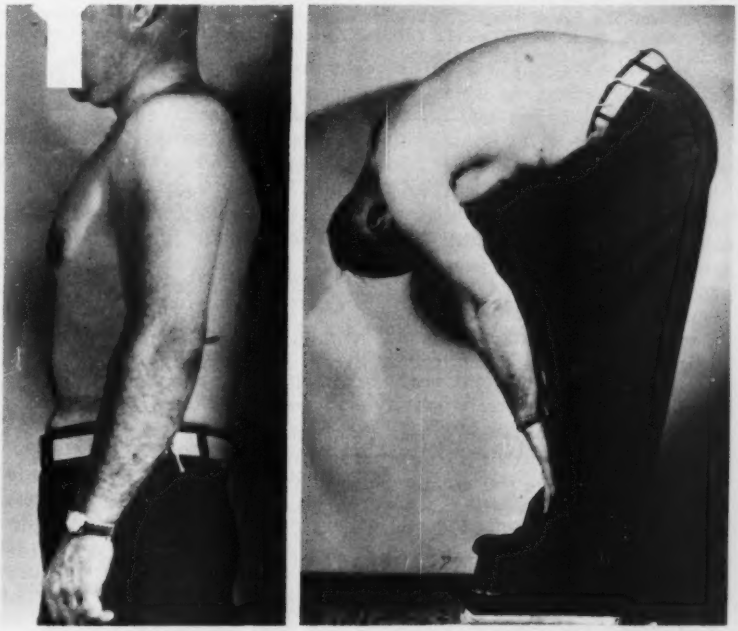

Figure 6. Appearance of patient and range of motion attained following treatment.

SUMMARY

The established teachings of the past masters of orthopedic surgery in the management of compression fractures of vertebrae remain unchallenged. In prevalent practice these fractures are treated for varying periods of time in hyperextension casts and back braces without reduction. Although the over-all results are good, significant residual complaints of a sociosomatic nature occur. Such complaints from back injuries are believed to arise in direct relationship to the length of time of treatment. A method of treatment and our results, in which the time factor is substantially reduced and where functional results are excellent, are presented.

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REVOLUTION IN THERAPEUTICS

To look at the present frontiers of medicine it is necessary first to look backward, because the frontiers of today are not the same as the frontiers of yesterday. When I was a student, it was common to feel that patients were admitted to the medical wards to lie and rot. The great watchword was "masterly inactivity," and many were attracted by surgery because of its comparative air of activity and of its desire to thrust forward. Yet it is medicine which has undergone the greater change. Today the atmosphere of the medical wards has changed. We are in the grip of a revolution in therapeutics. For the polypharmacy of yore, with its emphasis on mixtures and nostra, has been substituted by a battery of active remedies—antibiotics, antispasmodics, hypotensive drugs, and steroids—so much so that I sometimes feel that it is my job to go round the ward taking patients off, rather than to prescribe, the various medicines which pour into their mouths, their veins, and their buttocks. For these new magical remedies differ from the old in one important particular; the old remedies were at least inert and did not therefore harm though they did little good; but the new ones are powerful drugs with powerful actions and must be used with discrimination lest the patient suffer rather than benefit.—C. H. Stuart-Harris: The Frontiers of Medicine. *The Lancet,* 30 August 1958.

A CONTROLLED PSYCHIATRIC STUDY OF MILITARY PERSONNEL AT SICK CALL

JAMES W. MAAS, *Captain, USAF (MC)*

MANY statements are made as to the large number of patients seen in a general practice of medicine because of emotional factors. Most of the published data concentrate on impressions as to the incidence of psychopathology, general methods of handling emotional illness, and the orientation of the medical public.[1-8] Allen and Kaufman[9] of the Lahey Clinic quantified their results as to the presence of psychopathology.[9] They found that an incidence of 27.2 per cent of patients were best given a psychiatric diagnosis. In addition, 13.4 per cent had a combination of emotional and physical illness (a dichotomy of nomenclature, not fact). In an attempt to check these findings, as to the attendance to a general physician and not a diagnostic clinic, the following study was undertaken on a population of patients seen in military sick call.

Specifically, the questions to be reviewed were as follows: Is a patient seen in a physician's office likely to be a repeater as compared with a control group? If so, why? What is the incidence of patients with a clear-cut psychiatric syndrome? How many patients are overreacting to the presence of minor organic ills, *i. e.*, "a cold"? Will a man who has just arrived in an overseas theater, or one who is about to leave, be more likely to seek out the physician, *i. e.*, a nonpunitive parent figure?

It should be noted that the military lends itself particularly well to studies of this type. Because of the centralization of records a patient's history of past illnesses and visits to a physician are easily available. This facilitates the horizontal view of the medical history as well as making control groups accessible.

METHOD

To answer the foregoing questions, data were collected while seeing patients at sick call. Eighty consecutive patients were originally seen for this study. Controls were not obtained for four of the patients observed. Therefore, only 76 patients of the original 80 were included in the study. They were not aware the author

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was a psychiatrist. The sampling was random for, in our sick call, the patients were assigned to the physicians by the corpsmen who picked up the charts and called out the name of the patient without first seeing him thereby eliminating a conscious or unconscious selection on the part of the corpsmen. The following routine was followed with each patient. Past and family history was obtained and then, by use of the "associative anamnesis," data were collected as to the nature and severity of any symptoms of anxiety, a character and behavior disorder, or emotional factors which had prompted the patient to come to the physician. At the end of the interview the examiner directly inquired as to the presence of symptoms of anxiety not mentioned. The patient was then classified as follows:

A. Anxiety, acute and/or chronic:

0 - None:	No appreciable anxiety.
1 - Mild:	Very mild symptoms of anxiety.
2 - Moderate:	Symptoms of sufficient severity to cause the patient frequent and acute distress, *i. e.*, insomnia, tenseness, palpitations, nail biting, sweating palms.
3 - Severe:	Symptoms of such severity that the patient was at times unable to function. These were the patients who had histories of panic, hyperventilation, very low frustration tolerances, et cetera.

B. Presence of a character and behavior disorder:

0:	None noted.
1:	Very occasional and mild episodes of "acting out."
2:	To such a degree that their relationships with others and their superiors were threatened.
3:	These were patients with a history of one or more company punishments, courts-martial, demotions for disciplinary reasons.

C. Opinion of the examiner as to the emotional factors prompting the sick call visit:

0:	None apparent.
1:	Slight suggestion.
2:	Much evidence.
3:	No organic illness found and the patient presented a primary psychiatric syndrome.

The patient's record was then reviewed; it was noted how long he had been in Alaska and the number of months remaining prior

to leaving. The number of visits to sick call per three-month time interval was noted. Return visits at the suggestion of the physician were not included. Thus, only visits of the patient's own volition were counted (fig. 1).

MEDICAL SURVEY FORM

Last Name, Doe,	First Name John	Rank: A/3C Age: 18 Race: W Phone:	MARITAL STATUS Single: Married: Yes Divorced: Wife here: No	EDUCATION Grammar: 8 High School: 2 College: 0	Months in Service: 12
Organization 1882nd I & M, AACS					Months to Rotation: 14

Months in Alaska	0-3	4-6	7-9	10-12	13-15	16-18	19-21	22-24	25-27	28-30	31-33	34-36	37 & over
Visits to Clinic	1		3	4		1							

Clinical Data

Diagnosis:

Low back pain (sacral region). Physical exam and films are negative. Diagnosis: Psychophysiologic musculoskeletal reaction vs conversion reaction. Opinion as to emotional factors prompting visit: CLASS 2

Are there obvious NP overtones? (elaborate if yes)

Patient has acted out on several occasions since in the service

1) Suicide gesture
2) Increased drinking since assignment to area
3) Disciplinary demotion
4) Difficulty in high school; joined USAF

DEGREE OF ACTING OUT: SEVERE, CLASS 3

Does patient have symptoms of anxiety? (if so, which ones?) (if chronic, are they worse?)

Yes, palpitations, sweating palms, shortness of breath, restless, insomnia, feels tense. Symptoms have been present for two years. Acute increase in symptoms are coming more frequently.

ANXIETY ——— ACUTE AND CHRONIC MODERATE SEVERITY ——— CLASS 2

Is this a recurrence of an old illness? (if so, explain)

In 1952 fell off auto and injured (?) low back. In March 1956 was seen for paravertebral pains.

Miscellaneous

Figure 1.

A request was then sent to the patient's organization for the name of a man of similar age, race, rank, and level of education. This person's record was pulled from the hospital files and data as to the number of visits to sick call per three-month period were recorded. In addition, the number of months remaining prior to leaving this overseas station and months served were noted. In this way a control group was obtained which indicated the average number of visits per unit time, and influence of time spent in the area on sick call attendance. This group is considered to be a random sample of the organizations from which the patients came. In the strictest sense, this random sample

is not a *control* group nor is the other truly an experimental group. Nevertheless, economy of writing and common usage warrant the use of the terms *control* and *experimental*. With the foregoing in mind, these terms will henceforth be used. Because the control group was not individually interviewed, it was not possible to classify its members as to anxiety.

RESULTS

As can be seen from figure 2, there is a marked difference between the group representing a random sample seen in sick call and a control series. It would seem from this graph that a man seen in sick call in general will have nearly twice as many

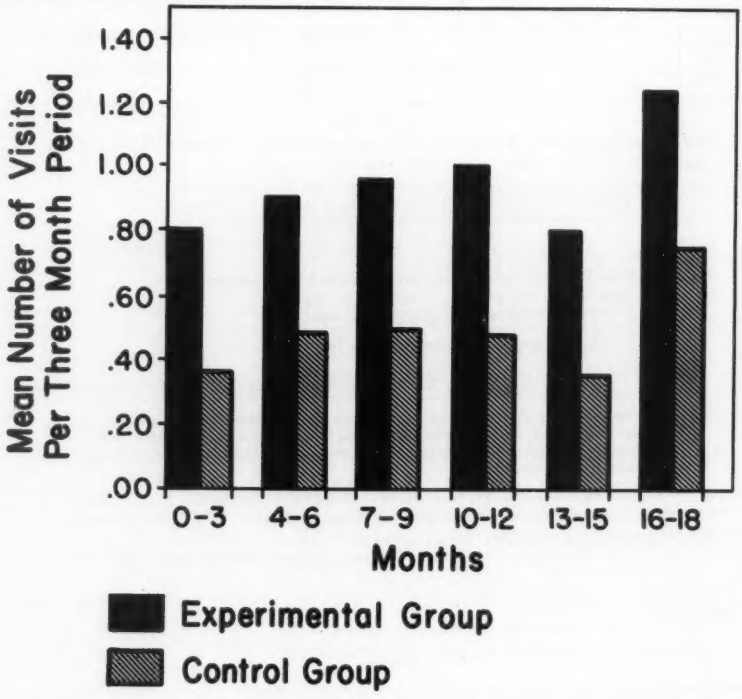

Figure 2. Comparison of experimental and control groups as to sick call attendance.

visits as a man in the control series. This is noted during all time intervals. In addition, it is interesting to note that the experimental group had a gradually increasing number of visits to the physician as the tour of duty out of the United States lengthened in time. This is most evident in the 16 to 18 months' period. These figures verify the clinical impression that persons who are seen by the physician are not a representative sample of the population, but, in fact, are a special segment.

Analysis of the data by the chi square method indicates the above difference cannot be ascribed to any significant difference in the number of men present for the various three-month periods in the two groups. In addition, the two groups were found to not differ as to the time in the theater at a statistically significant level.

Reviewing the clinical material of the experimental group, it was found that 32 per cent of the patients studied had been given a primary psychiatric diagnosis, whereas, 43 per cent had acute and chronic anxiety considered to be of pathologic proportions, *i. e.*, in the moderate to severe category. Most of these patients were seen, not for symptoms of anxiety, but for various ills such as upper respiratory infections (table 1).

TABLE 1. *Primary psychiatric diagnoses and anxiety in the experimental group (76 patients)*

Diagnosis	Number of patients	Per cent
Primary psychiatric:		
Conversion hysteria	9	
Anxiety reaction	5	
Gastrointestinal reaction	6	
Angioneurotic urticaria	1	
Musculoskeletal reaction	3	
Total	24	32
Anxiety of chronic type and sufficiently severe to be called pathologic:		
Moderate anxiety	21	
Severe anxiety	12	
Total	33	43

While the above data indicate a definite difference in the two groups, they in no way "prove" what is responsible for the variations. Inasmuch as it was the examiner's impression that one of the possible factors in sick call attendance might be the degree of psychopathology present, it was decided to break the experimental group into two separate categories. Those patients with moderate to severe anxiety, pathologic acting out, and/or a primary psychiatric diagnosis were compared to those who were not believed to have significant psychopathology. They were compared for three-month intervals up to the eighteenth month. The individuals in the groups beyond that period were not of sufficient number to be meaningful (fig. 3).

It is easily seen that those individuals who were classified as having psychopathologic conditions have a consistently higher number of visits than the group thought to be without significant psychopathology.

DISCUSSION

The above figures indicate that the sick call population, as a whole, differed from the control or randomly selected group. This might mean the patients seen were generally more prone to disease. This, of course, is a possibility. On the other hand, the high rate of psychopathology among the experimental group and, more importantly, the statistically significant association between psychopathology and the number of visits to the physician, suggest many patients are motivated to seek out a physician because of unresolved emotional conflicts. The format may be provided by some relatively minor illness such as a mild upper respiratory infection. This would account for the frequent

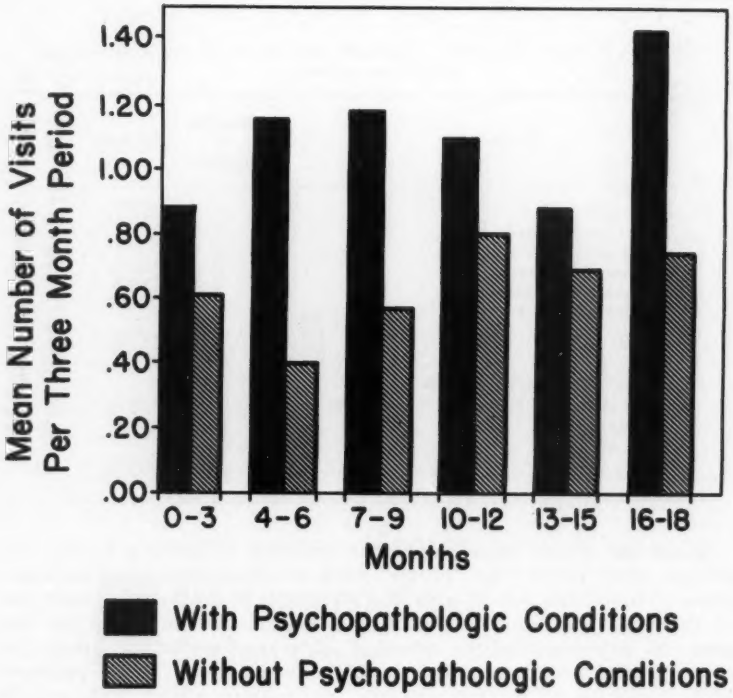

Figure 3. Comparison of sick call attendance of those with and without psychopathologic conditions. The data graphically presented were tested statistically in the following manner. The number of visits per three-month period (for total time in the area of assignment) was computed for each person in both groups. The figures thus obtained were handled via

$$t = (\bar{x}_1 - \bar{x}_2)\sqrt{\frac{n_1 n_2 (n_1 + n_2 - 2)}{(n_1 + n_2) x_2}}$$

The P value was less than 0.05, which is statistically significant.

observation that symptoms are often out of proportion to the illness. In addition, it may explain why many patients consult a physician with an illness of no consequence which past experience should have indicated to them is self-limiting. The patient, by attending sick call, might be using his symptoms as a defense against anxiety and, at the same time, making contact with a parental figure in an attempt to assuage his emotional discomfort. An interesting speculation along somewhat different lines is that emotional problems and resultant anxiety may predispose a patient to virus and bacterial infections.

When talking with patients, it was noted that their visits were occasionally cyclical. A person might be seen five times in a three-month period and then not again for many months. The associative anamnesis frequently indicated that the patient was undergoing some personally threatening situation at the time the increase in sick call visits was noted.

It was especially interesting to observe those patients whom many nonpsychiatric physicians would have classified as "gold brickers." This term frequently is put forth informally to explain the repetitive sick call visits by some patients. In this study, rather than an *explanation* of behavior, this was a *symptom* of a severely disordered personality structure. These patients frequently had anxiety and "acting out" of incapacitating proportions.

One of the original impressions prompting this study was that the sick call group would tend to have an initial increase in visits shortly after arriving in this overseas theater as a concomitant of separation anxiety. These data do not bear this out, but do show a trend toward increasing sick call attendance per three-month period with greater time in the area. When visits per unit time are graphed (fig. 2) it is found that there is a sharp upsurge in sick call attendance by both the experimental and control groups during the 16- to 18-month period. This might possibly be on the basis of impending reassignment to duty in the United States in 6 to 8 months, *i. e.*, separation anxiety due to expectation. If this were so, one would expect that increased attendance might be present during the succeeding time intervals. As mentioned earlier, the number of men present after 18 months do not allow a definitive answer. Because of the manner of selection of the material climatic conditions cannot be the etiologic factor.

Although this study was made on a military population, the findings may be directly applicable to a civilian practice of medicine. This is partially borne out by the similarity between the incidence of primary psychiatric diagnosis in the Lahey Clinic study,[9] 27.2 per cent, and in this one, 32 per cent.

Furthermore, it is important to note that the visit of a patient to a physician is an overdetermined act and as such one is not dealing with a randomly selected individual. This has direct pertinence to the way in which control groups in medical studies are chosen.

For example, one frequently sees a control series composed of a group of patients from a hospital ward differing from the one under investigation. In certain projects this may be adequate, but data from this study would indicate that in many instances (especially psychiatric research) such a practice could distort data and results due to the automatic bias introduced by using non-project-involved patients as controls.

SUMMARY

The frequency of visits to military sick call, and the occurrence of psychiatric disorder in 76 consecutive military personnel coming to sick call, were studied. The same data were analyzed in a matched group of airmen at the same military base. The military personnel seen in sick call had approximately twice as many visits per unit time as a matched, random sample of the base population. When the sick call group was categorized, there was found a statistically significant ($P = <0.05$) association between psychopathologic conditions and increased sick call attendance. Of the patients seen, 32 per cent had a psychiatric disorder accounting for their attendance to sick call. A total of 43 per cent had anxiety of a chronic and pathologic type. Most individuals in this latter group presented themselves with minor organic ills. It is concluded that emotional disturbances are not only associated with, but in a large part motivate, increased attendance at sick call.

ACKNOWLEDGMENT: The author is grateful to Col. Emmert C. Lentz, USAF (MC), 5040th U. S. Air Force Base, for his co-operation; to Comdr. Benjamin S. Gantz, Jr., USN, Elmendorf Air Force Base, for assistance on methods of statistical analysis; and to Drs. Louis A. Cottschalk, Stanley M. Kaplan, W. Donald Ross, Chester Pierce, and Goldine Gleser of the Department of Psychiatry of the University of Cincinnati.

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MALARIA AMONG TROOPS IN THE FIELD

Clinical Experiences in the Far East

CONSTANTINE J. SAMIOS, *Lieutenant, MC, USNR*

MALARIA was prevalent on Okinawa after World War II. Subsequently, however, preventive medicine units succeeded in almost eradicating the reservoir of the disease, and in the two years preceding this study only one case was reported to have originated among the military on Okinawa.[1] An anopheline mosquito population was present but malaria-free during this time.[2] There are many nearby areas in which endemic malaria is well recognized, however, and a constant flow of civilian and military traffic occurs between these and Okinawa. These include (1) the neighboring islands of Yaeyama and Ishigaki of the Ryukyu group, which maintain considerable commercial traffic with Okinawa; (2) Formosa and South Korea, with which a constant interchange of military personnel takes place; and (3) the Philippine Island of Luzon, which has been used as a maneuver area by the Third Marine Division for the past few years.

Since the fall of 1957, the Third Marine Division has held two training maneuvers on the island of Luzon: Operation Phiblink, involving a reinforced regiment of about 5,400 men who took part in ground maneuvers from about 29 November 1957 to 8 December 1957, and Operation Strongback, involving the entire Third Marine Division with about 10,000 men participating from about 27 February to 8 March 1958. In addition to these, a base camp of about 500 men, consisting of an engineer company and attached units, was maintained from 27 October 1957 to about 17 April 1958. The maneuver took place in central Luzon in the area of Dingalan, the Coronel River Valley, and Laur Cantonment Area, all of which are considered to be endemic for both *Plasmodium vivax* and *Plasmodium falciparum*. In the civilian population, the incidence of vivax and falciparum malaria are approximately equal. The primary vector is the mosquito *Anopheles (myzomia) minimus flavirostris*.

While in the Philippines, all the participating military personnel were required to take the following antimalarial precautions:

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1. Chloroquine phosphate, 0.5 gram weekly, as malarial suppressive, starting one to two weeks prior to arrival on Luzon and continuing for two weeks after departure. This was given by roster, usually on Sunday.

2. Mosquito nets. Base camp personnel were to use a full mosquito net nightly. For tactical reasons, most of the infantry units were unable to carry full-sized nets, but were supposed to use head nets after dark, with buttoned long-sleeved shirts and bloused trousers. In all, about 40 per cent of the men used full nets each night.

3. Mosquito repellent, to be applied every three to four hours after dark.

4. Insect spray, to be used inside tents and closed spaces.

In general, co-operation with these recommended precautions was as good as the tactical situation permitted. A temporary break in malaria control occurred following Typhoon Kit in November 1957, during which almost all the personal equipment of the men in base camp was lost. A one-week lapse in chloroquine suppression for most of the men followed, and most were without full mosquito nets for about one month afterward. Another break in control occurred when men stayed overnight in a native village.

The following study was made in Okinawa and consists of the presentation of clinical data on 47 consecutive cases of malaria occurring between 1 March 1958 and 15 May 1958. The only case on Okinawa in 1958 before 1 March was a recurrence of chronic malaria contracted elsewhere. After 15 May, in spite of 69 consecutive negative peripheral blood smears from men selected at random, the entire Third Marine Division was treated with a therapeutic course of chloroquine and primaquine phosphate to preclude the possibility of latent infections. This consisted of one dose of chloroquine, 1 gram, and daily doses of primaquine, 26.5 mg (15 mg of base), for 14 days by roster. In the past few years, most cases of malaria treated on Okinawa occurred during the period covered by this study.

Due to the field conditions under which this study was made, some difficulties were encountered. A control group in which chloroquine suppression was not used was impractical. No attempt could be made for long-term follow-up for recurrent parasitemia or clinical recurrences. Blood levels of chloroquine could not be determined. Most of the data was obtained by clinical interviews of the patients, with the inherent but unavoidable risk of inaccuracies in the patients' memory. This was reduced to a minimum by immediate interviews and by an explanation of the study to the patient to achieve the best possible co-operation.

CLINICAL MATERIAL

During the period of 1 March to 15 May 1958, 45 patients with vivax malaria and 2 with falciparum malaria were treated at Ryukyus Army Hospital. In 45 of these, the identification of the plasmodia was made by the laboratory of the hospital. Thirty-

nine of the 47 patients were personally interviewed and examined by the author.

In all cases, the disease was clinically mild and early. Six patients had not been in an endemically malarial region other than Okinawa for at least one year and had no past history of malaria. One patient had a history of what apparently was malaria, contracted in Korea. Forty, the remainder, had been in Luzon, Philippine Islands, for a period of time varying from 6 to 157 days, averaging 64 days. In one case the first malaria attack occurred only 14 days after arrival on Luzon; in another case it occurred as late as 191 days after arrival in Luzon and 113 days after departure from the area. No significant mean incubation period could be arrived at.

Typically, the patient had a history of malaise and bifrontal headache, worse with exertion and relieved temporarily or not at all by acetylsalicylic acid. This was followed by the development of chilliness and fever. Frequently, gastrointestinal symptoms were present, ranging from nausea or anorexia to abdominal pain and cramps, and loose stools or frank diarrhea (table 1). The chills were seldom "shaking" in character and myalgia was common. In only a very few cases was a definite time pattern of chills and fever established, inasmuch as medical attention was sought quite promptly, usually with the first attack.

TABLE 1. *Clinical findings in 45 cases of vivax malaria*

Symptoms or signs	Number of cases	Per cent
Subjective fever	41	91
Malaise and myalgia	40	89
Subjective chilliness	37	82
Headache	32	71
Gastrointestinal symptoms	23	51
Splenomegaly	13	29
Elevated serum bilirubin (1 mg per 100 ml or over)	11	24
Concurrent illness at onset	17	38

In 17 of the patients (38 per cent) the onset of clinical malaria was preceded for about 48 hours by another illness (table 2) which apparently occurred during the quiescent, latent phase and triggered the acute attack. In only 13 patients (29 per cent) was the spleen palpable.

Upon establishment of the diagnosis of malaria, treatment was started immediately. For vivax malaria, this consisted of chloroquine, 1.0 gram immediately, 0.5 gram in 6 hours, and 0.5 gram daily for three doses. Concurrently, primaquine, 15 mg of base, was given each morning for 14 days. Most patients continued to be febrile for 12 to 48 hours after start of the treatment, some of them exhibiting the most severe fever of their hospital stay at that time. All improved promptly thereafter and felt remarkably well in a very few days. The duration of hospital-

ization had no relation to the clinical severity of the illness. In most cases the final doses of primaquine were taken while the patient was back on active duty.

TABLE 2. *Incidence of concurrent illness in 17 out of 45 patients with vivax malaria*

Concurrent illness	Number of cases	Per cent of all cases of vivax malaria
Infectious mononucleosis	5	11
Epidemic diarrhea*	4*	9
Respiratory tract infections*	4*	9
Urticaria	2	4
Genitourinary tract infections	2	4
Inoculation reaction	1	2

*One patient had acute coryza with diarrhea at onset of malaria.

CHLOROQUINE SUPPRESSION

Chloroquine phosphate has been accepted as the malarial suppressive of choice for troops operating in endemic areas. It is administered orally as a single 0.5 gram enteric-coated or plain tablet weekly, achieving peak blood levels of 150 to 250 micrograms per liter. It is stored in the lungs, liver, kidney, and spleen, and excreted quite slowly so that after one week the blood level is still 20 to 40 micrograms per liter.[3] For treatment of acute attacks, a level of 5 to 8 micrograms per liter is reportedly adequate. According to standard texts,[3] after an initial two doses, chloroquine administered once weekly is supposed to protect against the development of overt malaria from "several weeks" to "many months" after the last dose. In our series, however, 50 per cent of the patients developed their first symptoms of malaria less than 24 days after taking their last chloroquine (fig. 1), a period of time during which they should have had ample blood levels to continue to suppress the malaria. Eight patients developed their first symptoms while taking the drug, less than one week after their last dose.

Chloroquine, in the dosage given, does not completely suppress this form of vivax malaria. This may have been due to inadequate absorption from the gastrointestinal tract. It also is evident, that chloroquine does significantly decrease the incidence of malaria, as shown by the low over-all incidence of only 47 cases in a population of 10,000 men engaged in training simulating combat in an infested malarial region. It also is shown by the extremely low incidence of falciparum malaria—only two cases, one of which was in a patient who had taken his chloroquine only erratically. According to medical intelligence reports[4] the incidence of falciparum and vivax malaria is approximately equal in the concerned areas, so apparently a large number of cases of falciparum malaria, which is potentially the most deadly, may have been prevented.

CONCURRENT ILLNESSES

It has been noted that 38 per cent of the patients were admitted with initial symptoms due to disease other than malaria (table 2). The most common condition occurring concurrently was infectious mononucleosis, present in five patients (11 per cent). This inci-

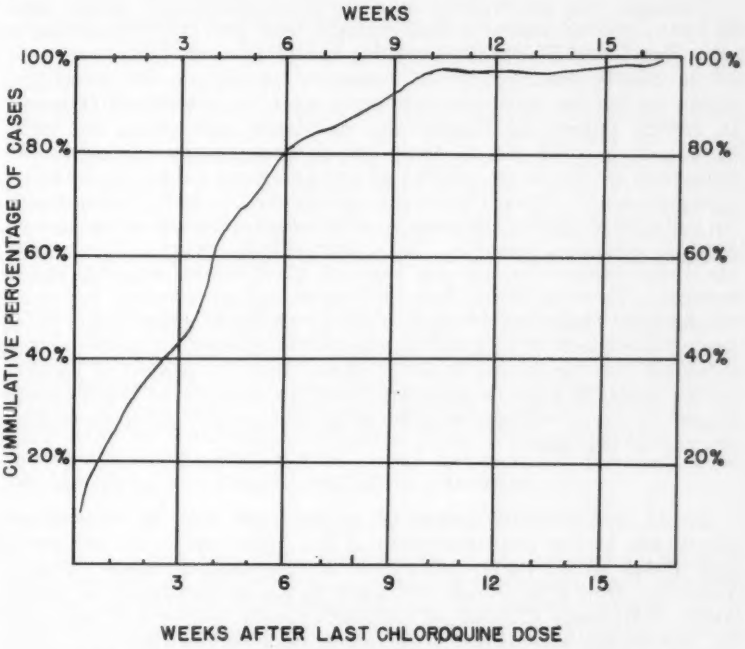

Figure 1. Onset of vivax malaria after chloroquine suppression.

dence is much higher than would be expected and it may be speculated that the presence of this viral infection in the reticuloendothelial system lowers the resistance and hastens the clinical onset of the *Plasmodium vivax* infection from this system, or vice versa. No significant difference in efficiency of chloroquine suppression could be shown in the cases with concurrent illness.

During the months this study was in progress an island-wide epidemic of diarrhea broke out. Four patients with diarrhea subsequently developed overt malaria. No etiologic agent for the diarrhea was incriminated, but it is certain that the diarrhea epidemic was not due to gastrointestinal (algid) malaria because of the large number of patients with diarrhea but without malaria. Again, it is believed that the acute illness merely triggered an existing latent plasmodium infection into the full-blown clinical illness.

It is noted that two patients had urticaria (giant hives) on admission. No specific allergen was subsequently identified and it may be postulated that the atopic reaction was due to the foreign protein in the plasmodium parasite, such as frequently occurs with trichinosis.

OKINAWA AS AN ENDEMIC AREA

Although only incomplete data are available for the years 1945 to 1955, reports indicate that malaria was prevalent on Okinawa after World War II. By 1952, public health measures had succeeded in nearly eradicating the reservoir so only a few scattered cases among the military population have originated on Okinawa in recent years. No figures are available concerning the incidence, if any, in the civilian population. However, due to the existence of the large number of the potential vector *(Anopheles hyrcanus sinensis)* on the island and the heavy traffic with close-by malarial regions, the danger of re-establishment of the reservoir on Okinawa remains. This year (1958) malaria occurred in six individuals who had not been in other known malarial areas recently. Three of these definitely have had no previous exposure to malaria. Three others have been in malarial areas in the past, but on the basis of clinical evidence are thought to have become infected on Okinawa. None of these had symptoms of malaria in the past. It also is possible that one or more of the 38 other cases of vivax malaria attributed to Luzon may have been contracted on Okinawa.

SUMMARY AND CONCLUSION

In 47 consecutive cases of malaria the use of chloroquine phosphate in the dose schedule of 0.5 gram weekly is evaluated and found to be partly effective in suppressing vivax malaria. However, this effect does not seem to be as prolonged as previously indicated. Attacks of clinical malaria were often triggered by incidental infections. The occurrence of six cases of vivax malaria apparently originating on Okinawa is noted as evidence that malaria is once more threatening to become a significant hazard to military personnel there.

ACKNOWLEDGMENT: This study was made as part of the Third Marine Division Preventive Medicine Unit under Captain J. M. Amberson, MC, USN, and could not have been possible without the help and co-operation of Captain Amberson and W. E. Pierce, HMC, USN.

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ROENTGENOGRAPHIC MANIFESTATIONS OF PULMONARY SARCOIDOSIS

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JAMES M. KEEGAN, *Lieutenant Colonel, USAF (MC)*

SARCOIDOSIS is a disease of unknown cause which can affect any organ or system of the body but appears to have a special predilection for the lungs and their lymphatic drainage areas. Pulmonary sarcoidosis usually is a fairly benign process which runs its course with few, if any, symptoms, and undoubtedly is unrecognized in many individuals. Occasionally, however, its manifestations can be troublesome and its outcome serious.

Although the majority of cases of pulmonary sarcoidosis present little problem in diagnosis, there are some in which diagnosis is difficult because of minimal or bizarre roentgenographic appearances. During the past five years, 48 histologically proven cases of sarcoidosis were seen at this hospital. Of these, 40 had thoracic involvement of some type. Roentgenograms of the chest were available for study in 38 cases which form the basis of this report.

CLINICAL FEATURES

The age, sex, and race distribution of this series is of little significance in the over-all evaluation of sarcoidosis because of the high degree of selectivity of our patient population. It has been well established, however, that sarcoidosis is most commonly found in the 20 to 35-year-old age group, is more prevalent in females than in males, and in this country shows a much higher percentage incidence in the Negro than in the Caucasian population.

The age range of our patients was from 17 to 39 years, with a mean age of 24 years. Of our patients, 35 were males and three were females; 16 were Negroes.

Of the 38 patients, 11 had symptoms at the time of discovery of their disease. In only five of these, however, were the symptoms significant enough to cause them to seek medical attention. Dyspnea was the chief complaint of one patient; two had a nonproductive cough, and two had cough, dyspnea, weakness, and weight loss. The other six patients with symptoms volunteered the information when questioned that they had had an intermittent

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dry, hacking cough. It was interesting to note that at the time of discovery of disease, 27 cases were completely asymptomatic, and 33 were discovered on routine chest roentgenograms. This disease often remained asymptomatic throughout its course, even though the chest roentgenographic findings were quite marked.

PATHOLOGIC MATERIAL

The most successful method of histologically identifying this disease in our cases was by scalene node biopsy. Thirty-three were proved by scalene node examination, two by liver biopsy, one by skin biopsy, one by lung biopsy, and one by both scalene and liver biopsy.

Microscopically, sarcoidosis is characterized by the presence of epithelioid cell tubercles in a tissue or organ. These tubercle-like lesions have little, if any, necrosis and eventually undergo regression by fibrosis. The fibrous tissue formed in the wake of the tubercle rapidly becomes compact and hyalinized with the eventual development of dense scar tissue.[1]

ROENTGENOGRAPHIC FINDINGS

The roentgenographic appearance of pulmonary sarcoidosis is dependent on the stage of the process and the extent of involvement. The usual course of the disease is characterized by a fairly definite pattern of involvement of thoracic structures. The varying roentgenographic manifestations found initially result from discovery of the disease in one of its several phases.

Enlargement of the hilar and paratracheal lymph nodes, with or without minimal lung involvement, is the first thoracic manifestation of sarcoidosis. This adenopathy may be quite striking in appearance. When the disease is seen in its formative stage, the nodes may show significant progression in size in a short period of time.

This enlargement of the hilar and paratracheal nodes usually regresses over a period of months or years, without roentgenographic evidence of significant lung involvement. Disseminated parenchymal disease subsequently develops in some cases which have node enlargement, and will usually appear as the enlarged nodes begin to decrease in size.[2] These parenchymal changes may regress, sometimes quite rapidly, or they may go into a chronic phase with the eventual formation of pulmonary fibrosis and emphysema.

MATERIAL

At the time of diagnosis, 16 of our patients had only enlarged nodes. In most of these patients, both of the hilar and both of the paratracheal areas were involved. However, any group or combination of groups of nodes were found to be enlarged. In our cases, the left paratracheal chain of nodes was involved as frequently as the right, but this was not as evident on the roentgenogram because this area is partially obscured by the aortic arch and pulmonary artery (fig. 1). One of these cases

had such extensive node enlargement that the nodes caused bronchial compression with atelectasis of the right middle lobe and a portion of the right lower lobe (fig. 2). This complication

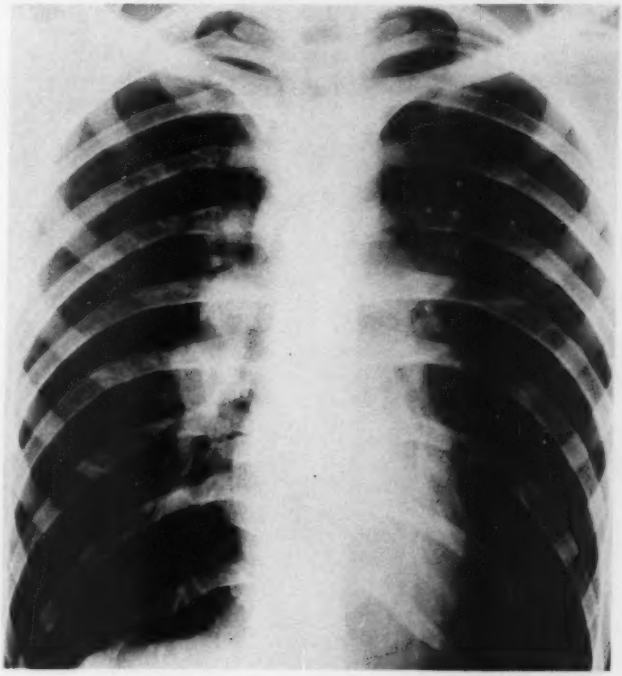

Figure 1. Sarcoidosis demonstrating bilateral hilar and paratracheal node enlargement. Note the enlarged left paratracheal nodes partially obscured by the aorta and pulmonary artery.

apparently is not common, as there are only two other cases of right middle lobe syndrome due to sarcoidosis reported in the literature.[3,4]

Five of our cases had enlarged nodes and some minimal lung involvement. This lung involvement consisted of a few small scattered patches of parenchymal infiltration and linear fibrous stranding, usually confined to the upper lobes (fig. 3).

We observed the development of pulmonary sarcoidosis in five patients, all of whom had normal roentgenograms of the chest within a 4-month period prior to the development of their disease. One of these was a known case of sarcoidosis with liver and ophthalmic involvement. When pulmonary disease developed in this patient, only the left paratracheal nodes became enlarged, remained large for several months, and then regressed. Two patients had node enlargement in all areas, and two devel-

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oped node enlargement and, simultaneously, some minimal parenchymal infiltration in the upper lobes. One of these latter patients was followed for three years. The nodes disappeared within a year, but the parenchymal lesions remained essentially unchanged for two years, and then gradually became tiny, fibrotic strands.

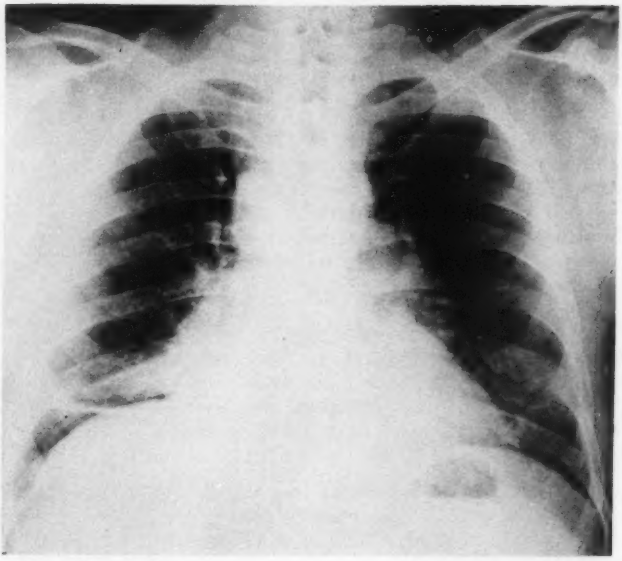

Figure 2. Bilateral hilar and paratracheal node enlargement are present. There is atelectasis of the right middle lobe and a portion of the right lower lobe.

Some form of generalized lung involvement was observed in 17 of our patients. Of these, 15 could be classified as having early or acute form of the disease, and two as late.

Of the 15 cases of early parenchymal disease, eight had definite node enlargement; one changed from predominant node involvement to lung involvement. The nodes receded as the parenchymal disease increased. This change took place within a 9-month period of time.

Three types or patterns of parenchymal disease were observed on the roentgenograms, although some intermixing of the types was present in most cases. A fine reticular pattern predominated in five cases (fig. 4). A coarse reticular pattern, usually accompanied by a few small coalescent areas scattered throughout the lungs, appeared in six cases (fig. 5). Large, patchy areas of increased density, with fluffy, irregular margins, were seen in three cases (figs. 6-8).

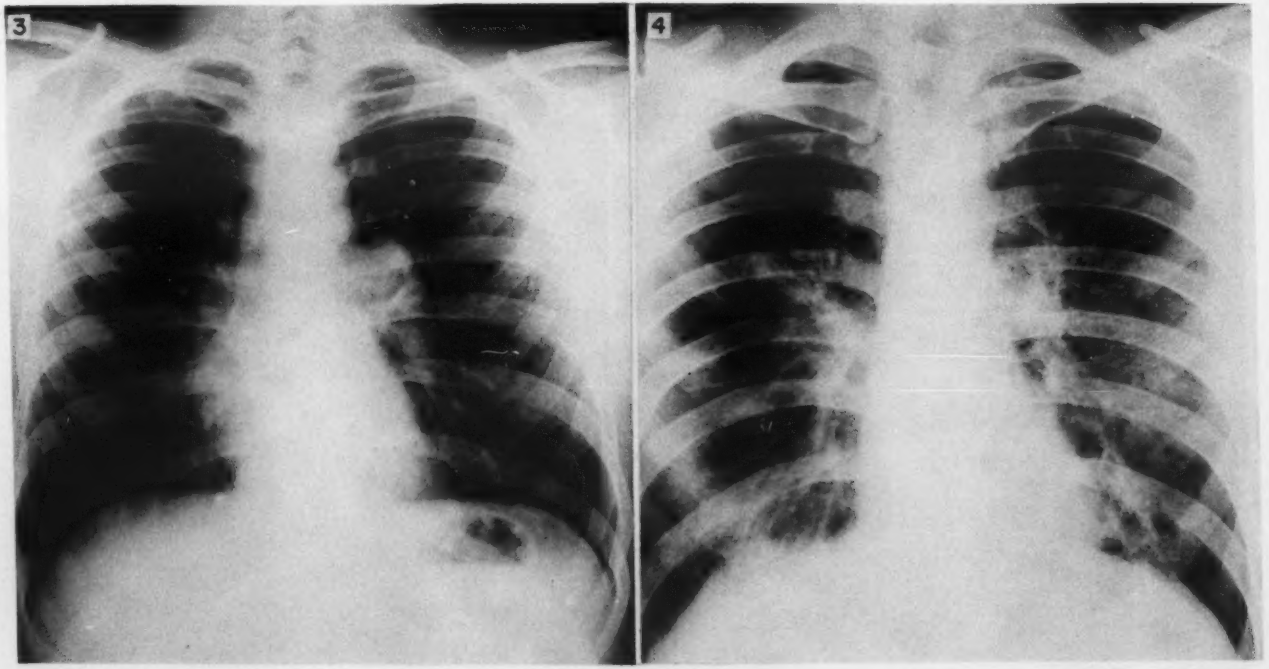

Figure 3. In this case, there was simultaneous development of hilar adenopathy and minimal parenchymal disease of the right upper lobe. Figure 4. Sarcoidosis with diffuse lung involvement manifested as a fine reticular pattern.

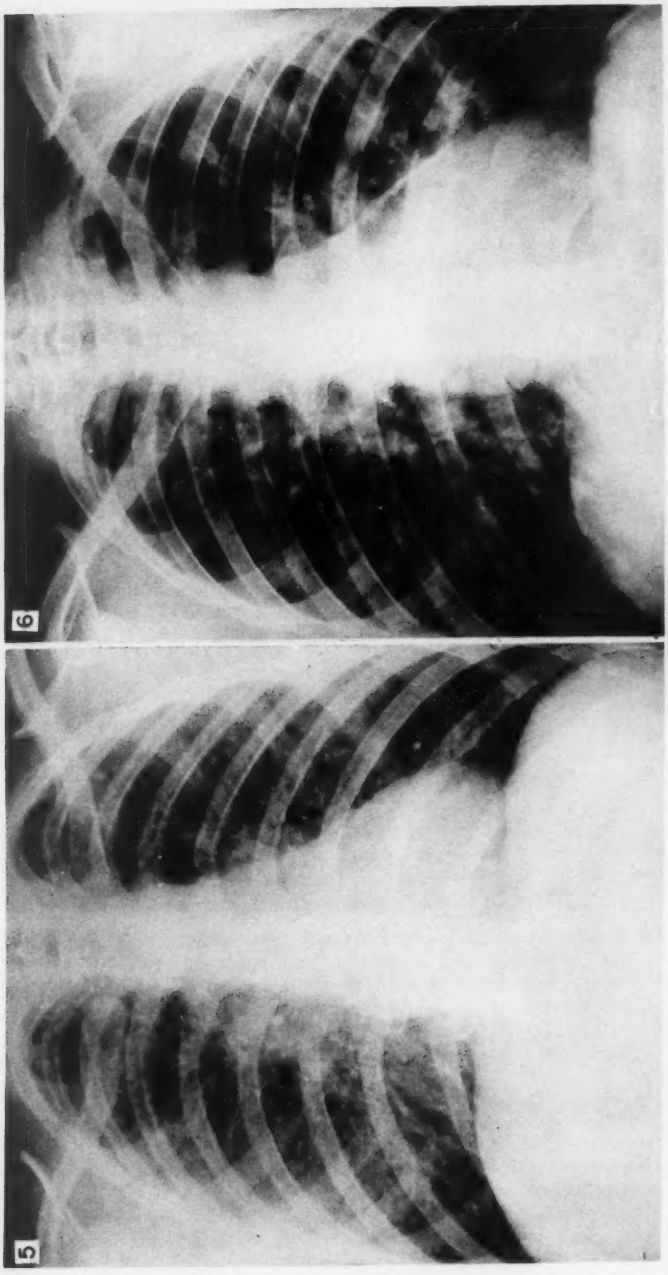

Figure 5. Sarcoidosis with a coarse reticular pattern throughout both lungs. Figure 6. Sarcoidosis showing intermixing of a coarse reticular and a patchy nodular infiltrate in both lungs.

Figure 7. This case demonstrates sarcoidosis with a nodular type of lung involvement. Hilar and paratracheal node enlargement are present. Figure 8. Same case as figure 7. Two months later, there had been almost complete resolution of the entire disease process. Note the presence of intrapulmonary pleural effusion on the right. Two patients in this series developed pleural effusion.

The late stage of this disease is characterized by the occurrence of extensive fibrosis and emphysema (fig. 9).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of sarcoidosis can be divided into two main groups. Those cases with hilar and paratracheal adenopathy must be differentiated from the adenopathy found in many diseases, but the most important differential is between sarcoidosis and lymphoma. Sarcoidosis uncommonly involves the nodes of the anterior mediastinum, whereas the lymphomas often

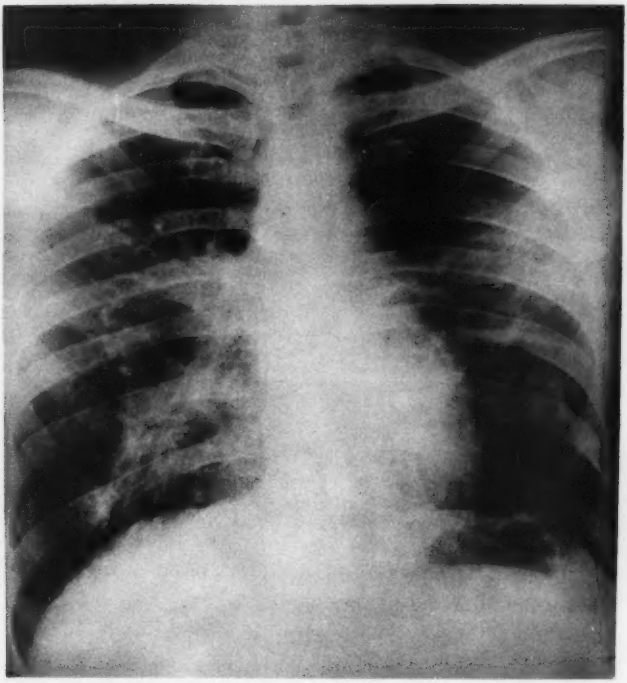

Figure 9. This case demonstrates the late changes which occur in pulmonary sarcoidosis. There are extensive fibrosis and emphysema throughout both lungs.

do.[5] Adenopathy in the anterior mediastinum can be identified on the lateral roentgenogram of the chest. Lateral chest roentgenograms were available for study in 36 of our 38 cases. In no instance were the nodes in the anterior mediastinum enlarged, although in two cases the paratracheal adenopathy was of such magnitude that it appeared to encroach on the anterior mediastinum.

When parenchymal involvement is present, with or without node enlargement, sarcoidosis must be differentiated from tuberculosis, coccidioidomycosis, berylliosis, Hamman-Rich idiopathic

pulmonary fibrosis, alveolar cell carcinoma, and pneumonia, depending on the pattern of involvement in each particular case.

SUMMARY

Pulmonary sarcoidosis is a disease which undoubtedly occurs more frequently than is generally recognized. It is frequently asymptomatic and is often discovered on routine roentgenographic examination of the chest.

Pulmonary sarcoidosis appears to follow a fairly definite pattern of involvement of thoracic structures. The initial involvement is manifested on the roentgenogram by enlargement of the hilar and paratracheal nodes, with or without some minimal lung involvement. Extensive parenchymal involvement may occur as a later manifestation. This parenchymal involvement is seen in several patterns.

The entire disease process usually regresses over a period of months or years, but occasionally will go into a late or chronic phase characterized by fibrosis and emphysema.

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DEATHS FROM HARMFUL CHEMICALS

Nearly 8,000 persons in the United States lost their lives from accidental and intentional overexposure to harmful chemicals in 1955. These figures do not include deaths due to food poisoning, acute and chronic alcoholism, or poisoning by venomous animals. Over one half of the listed fatalities were classified as unintentional poisonings, that is, deaths were due to accidents, medical misadventure, and occupational exposures. . . .Since the major portion of all fatal accidental poisonings occur in or around the home, the higher incidence of injury due to medicinal chemicals is greatly influenced by the widespread casualness about the toxic properties of common drugs and easy access to those capable of causing harm.—Bernard E. Conley: Morbidity and Mortality From Economic Poisons in the United States. *A. M. A. Archives of Industrial Health,* August 1958.

THE SUBMAXILLARY GLAND IN NATURALLY ACQUIRED CARNIVORAL RABIES

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YUTAKA FURUSHO, *D. V. M.*

INVESTIGATION of rabies has continued since Pasteur's dramatic and classic experiments,[1] but advance in fundamental research deriving from fortuitous or acute observations has been meager. Important basic studies have dealt with evidence of the relationship of rabies virus and the parasitized host cell.

Hyden[2] reported that rabies elementary particles are self-reproducing units. They attack the nucleoprotein-forming center in the region of the Purkinje cell's nucleolus-associated chromatin. Particles rich in desoxyribonucleic acid are formed within the nucleus of the Purkinje cell of a rabbit infected with rabies. Ultraviolet light absorbing spots give a positive Feulgen nucleal stain. Electron microscopy has revealed characteristic vacuoles within Negri inclusions in hippocampal cells of a rabies-infected mouse.[3] However, study did not substantiate identity of virus elementary bodies. Recent cytochemical investigation of inclusion bodies of rabies[4] confirmed earlier observations[5] that Negri bodies do not give Feulgen-positive nucleal reaction and do not contain detectable levels of ribonucleic and desoxyribonucleic acids. The Negri body is neither simple carbohydrate, neutral fat, nor crystalline material; but is comprised of dry substance, possibly mainly proteins, in larger amount than in surrounding cytoplasm. Negri bodies appear approximately 3 days after demonstration of infective virus in animal tests.[6] The question of the origin of the Negri body is unknown although Negri inclusion bodies almost without exception occur after infection with natural or so-called street-virus rabies.[7] It is a conspicuous fact that the relation of the Negri body and rabies virus remains obscure. A great amount of work has been directed to the latter question, but the central nervous systems of infected animals have not yielded workable clues.

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The relationship of the Negri body and virus has proved of extreme complexity to resolve. It may be reasonable to speculate, therefore, that the preferential site of rabies virus development and maturation in the carnivoral host in natural disease has not been sharply identified.

The salivary glands, in contrast to brains of rabid animals, are noticeable for the comparative neglect that they have received in fundamental research. For example, the Expert Committee on Rabies of the World Health Organization[8] proposes a number of suggestions for future research. The basic research of salivary glands of naturally rabid carnivoral hosts is touched on slightly in connection with ecologic investigation. Neither histopathologic nor immunologic technics permitting the localization and visual demonstration of the rabies particle or distinctive pathologic biochemical products of infected salivary gland cells have evolved. An important fact is widely known concerning dogs naturally infected with street-virus rabies, namely, that the mandibular gland, on fresh weight basis, frequently contains more infective virus material than brain tissue of the same animal.[5,9-10] The virus associated with infected submaxillary glands of dogs has high stability and can be stored almost indefinitely at -70°C.[11] Experiments utilizing infected submaxillary gland suspensions as challenge material in rabies immunity research, therefore, have been conducted.[12-14]

This laboratory has instituted a long-term study of rabies virus in the mandibular salivary gland and brain sites of naturally rabid animals. Procedures that effect partial purification and concentration of rabies virus in material from the submaxillary gland have been explored. The characteristic property of the virus from mandibular salivary glands of rabid dogs has been studied. As a consequence of the work reported herein, microscopic corpuscular elements in infected submaxillary glands, not previously recognized, have been found.[15]

MATERIAL AND METHODS

Street-Virus Material. Materials infected with rabies virus were obtained from dogs in Korea with naturally acquired disease. Brain and submaxillary salivary glands were obtained from animals that died from gunfire or following a quarantine period. Wet impression[16] and histopathologic staining procedures were applied to brains of animals for detection of Negri bodies.

Intracerebral Titration of Virus. Ten and 20 per cent by weight of each of submaxillary salivary gland and brain were macerated in a Ten Broeck tissue grinder. The sterile diluent comprised M/150 Sorensen's phosphate buffer, pH 7.2, 0.85 per cent sodium chloride, and 0.63 mg (1,000 units) crystalline potassium penicillin G per ml. Coarse tissue fragments in the homogenate after disintegration in the Ten Broeck grinder, were removed by

centrifugation for 10 minutes at 1,600 r. p. m. in a Model PR-2 International Refrigerated Centrifuge. The supernatant was the conventional "10 per cent" or 10^{-1} tissue suspension. Tenfold serial dilutions of tissue homogenate were made in modified Sorensen's buffer solution with penicillin, containing in addition bovine serum albumin of about 100 mg per cent nitrogen concentration. Inoculums of 0.03 ml of each dilution of tissue suspension were injected intracerebrally in five mice. Animals were observed twice daily for 30 days. Titers of infectivity, expressed as negative log LD_{50} were calculated by the method of Reed and Muench.[17]

Nitrogen Determinations. Suspensions of brain and submaxillary gland tissue of dogs were sampled before and after treatment with Attasorb.* Nitrogen assay was achieved by a standard macro-Kjeldahl procedure.[18] Nitrogen was expressed in mg per 100 ml of tissue suspension.

Virus from Submaxillary Gland. The submaxillary salivary gland, situated at the posterior border of the dog's mandible, is elliptical in shape, and grayish yellow or orange in color. Histologically, it is a mixed type salivary gland, comprising physiologic groups of serous and mucus-secreting cells. The tough fibrous capsule encircling the gland was dissected away. The gland tissue was weighed and cut with sharp, sterile, curved scissors. The preliminary mincing rendered the gland tissue suitable for mechanical disintegration of the cell components. Five grams of the minced salivary gland were ground up manually with the aid of a 50-ml Ten Broeck grinder. Careful manipulation was necessary to avoid accidental breaking of the tissue grinder. A modified Sorensen's phosphate buffer solution was used as aqueous diluent. It contained Sorensen's M/15 mixture of monobasic potassium phosphate (KH_2PO_4), 28 ml; anhydrous dibasic sodium phosphate (Na_2HPO_4), 72 ml; and distilled water to 1 liter. Sodium chloride, 8.5 grams, was added. The final pH was 7.2. Penicillin supplement of 1,000 units per ml minimized bacterial contamination. Coarse tissue fragments were removed by low-speed centrifugation for 10 minutes at 1,600 r. p. m. The viscid opalescent supernatant was the starting "10 per cent" glandular tissue suspension. The viscosity noted was derived from the thick and slimy secretion of the mucous glands. The homogenate was treated with a chemically inert, natural-occurring siliceous material, described as Attasorb or Attaclay SF. Attasorb is a fine powder, of particle size of 400 to 600 millimicrons in diameter. It possesses high sorptive property for low molecular weight cellular constituents. It provided a mechanical trap for cell and nuclear walls and other extraneous tissue fragments. A quantity of sterile, autoclaved, dry Attasorb was added in proportion of 10 grams to 90 ml "10 per cent" gland suspension, the mixture shaken vigorously by

*Attasorb or Attaclay SF (now Pharmasorb) is a product of Minerals and Chemicals Corporation of America, Menlo Park, N. J.

hand, and centrifuged for 10 minutes at 2,500 r. p. m. at 5°C. The absorbent clay was discarded. The resultant aqueous phase has been termed Attasorb-treated supernatant. Attasorb possesses a rare ability to reject high-molecular weight, cellular-invading, infectious moieties such as virus[19] and complex, polymerized desoxyribonucleic acid.[20] In the mandibular salivary gland of the rabid dog, Attasorb failed to absorb submicroscopic granules and distinguishable microscopic elements.[15] The clay-absorbed, viscid-aqueous phase was centrifuged for 1 hour at 17,250 r. p. m. at 5°C, in an International Portable, PR-2, Refrigerated Centrifuge. The centrifugation yielded a clarified but still opalescent viscid-aqueous portion designated high-speed supernatant and a small pellet of gelatinous material identified as sedimentable fraction.

RESULTS

Nitrogen estimations of six preparations each of brain and submaxillary salivary gland are summarized in table 1. The presence or absence of rabies virus in these preparations was established by animal test. Attasorb treatment removed approximately 58 to 71 per cent nitrogen from 20 per cent brain tissue suspensions. The reduction in nitrogen with Attasorb of 10 per cent submaxillary gland suspensions varied from 31 to 78 per cent.

TABLE 1. *Nitrogen content of suspensions of brain (A) and submaxillary salivary gland (B) of dogs before and after adsorption with Attasorb*

Specimen	Per cent suspension	Total nitrogen[2] (mg/100 ml) Attasorb[3] treatment		Per cent N reduction	Rabies diagnosis by animal test
		Before	After		
1 A	20	115	38	67	Negative
1 B[1]	10	156	81	48	
2 A	20	129	42	68	Negative
2 B	10	138	31	78	
3 A	20	116	41	65	Negative
3 B	10	144	47	68	
4 A	20	122	36	71	Negative
4 B	10	122	84	31	
5 A	20	100	42	58	Positive
5 B	10	88	35	60	
6 A	20	127	43	66	Positive
6 B	10	158	87	44	

[1] Wet weights of salivary glands varied from 10 to 20 grams. Resulting suspensions were highly viscous.
[2] By macro-Kjeldahl procedure.
[3] Product of Minerals and Chemicals Corporation of America, Menlo Park, N. J.

Table 2 shows mice titers of infectivity of tissue suspensions of dogs which had acquired rabies disease naturally. Infective titers of pre-Attasorb treated brain and submaxillary gland suspensions are consistent with findings of other workers. Brain tissue suspensions, on fresh weight basis, contained tenfold to a hundredfold less measurable infectivity than submaxillary gland in mice tests. The titers of virus infectivity of *post-Attasorb* treated suspensions presented a different finding. Three treated preparations of brain suspensions failed to induce rabies in mice in 30 days. This may be interpreted to mean that adsorptive Attasorb reduced the virus level in the dog brain suspensions to less than a minimal infective dose. In contrast, Attasorb applied to submaxillary gland rejected the major proportion of the infective property of these suspensions.

TABLE 2. *Street virus in brain and mandibular salivary gland of rabid dogs*

Preparation	Mice titer of infectivity				Type of clinical rabies
	Brain		Salivary gland		
	Untreated	Attasorb	Untreated	Attasorb	
1	2.60[1]	n t[2]	3.64	n t	Furious
2	2.63	<1.0[3]	4.63	4.32	Furious
3	3.75	<2.0	5.50	4.50	Furious
4	3.10	<1.0	5.20	4.50	Furious
5	<1.0[4]	n t	<1.0[4]	n t	Dumb

[1] The infective titer of the tissue suspension is expressed as the negative log LD_{50}, 30 days following intracerebral inoculation.
[2] Not tested.
[3] No deaths observed in 30 days.
[4] Rabies virus isolated. Microscopic examination of original dog brain negative for Negri bodies.

The size and weight of infective particles in submaxillary gland suspension after absorption with Attasorb were investigated. High-speed centrifugation yielded a sedimentable pellet of gelatinous substance possessing measurable mice infectivity nearly indistinguishable from starting submaxillary gland suspension (fig. 1). The chemically inert, siliceous material had assisted in achieving a nearly hundredfold partial purification of sedimentable rabies infective material, as measured in terms of the nitrogen reduction shown in table 3.

A recapitulation of the composite experimental findings of pre- and post-Attasorb treated tissue suspensions of a case of naturally acquired disease may be seen in figure 2. The important points to note herein are (1) submaxillary gland suspensions induced rabies disease in rabbits inoculated intramuscularly after a relatively short incubation period; (2) infective brain suspensions, notwithstanding the fact that they contained a hundred-

fold less infective virus, failed to induce rabies disease during 75 days of observation; and (3) rabbits that died following inoculation with infective submaxillary gland showed good brain

TABLE 3. *Attasorb, chemically inert siliceous material, for nitrogen reduction of submaxillary gland suspension*

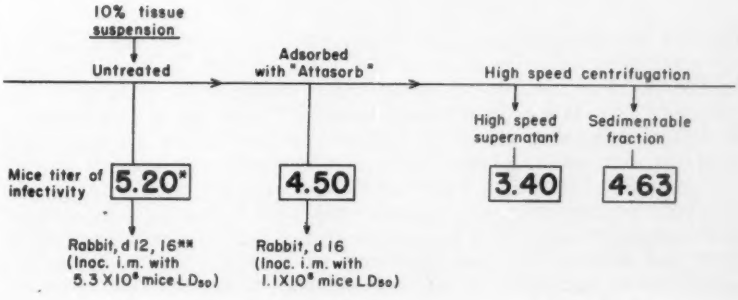
10% submaxillary gland suspension	Preparation number	Nitrogen[1] (mg/100 ml)		Remarks
		Total	% reduction	
Original pre-Attasorb	1	237	-	
	2	280	-	
Original[2] pre-Attasorb	3	120	-	Supernatant
Supernatant post-Attasorb	1	57	76	Adsorbed with Attaclay S F
	2	124	56	
	3	49	59	
High-speed supernatant, post-Attasorb	1	47	80	17,250 rpm, for 1 hour, at 5°C
	2	101	60	
	3	41	66	
Sedimentable fraction	1	10[3]	96	58-6-1, rabies pos.
	2	23[3]	92	58-6-6, rabies neg.
	3	8[3]	93	58-4-61, rabies pos.

[1] By macro-Kjeldahl procedure.

[2] It is conventional procedure to remove gross tissue particles by short-time, low-speed centrifugation.

[3] By calculation.

titers of infectivity, but their submaxillary salivary glands, prepared in conventional manner, that is, "10 per cent suspension," failed to yield detectable virus by animal test method.



* Expressed as the negative log LD_{50}, 30 days following intracerebral inoculation.

** Incubation in days to death of animal.

Figure 1. Concentration of street virus from mandibular salivary gland of rabid dog.

Microscopic corpuscular elements of submaxillary gland of a dog with naturally acquired rabies disease are shown in figure 3.

DISCUSSION

The technic of adsorption with inert siliceous Attasorb has achieved the separation of an apparently nonspecific nitrogen-complex salivary gland component, without noticeably altering the quality or infectivity and quantity of the rabies-disease-inducing moiety. Salivary gland tissue suspensions of dogs are characteristically viscous. Variations in reduction in nitrogen

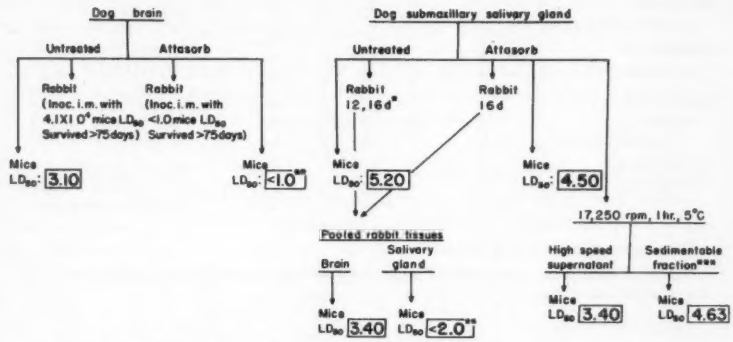


Figure 2. Property of virulence of street virus in 10 per cent tissue suspension

content of submaxillary gland component of 31 to 78 per cent may be attributed to a function of the viscosity of the preparations. In contrast, separation of nonspecific nitrogenous impurities has yielded infective post-Attasorb submaxillary gland suspensions of uniform property to incite rabies disease (table 2). Dog brain suspensions are relatively rich in virus material but are devoid of viscosity. It is not necessarily remarkable that reduction in nitrogen content of brain suspensions is accompanied by loss of detectable levels of the infectious component. This is consistent with the findings of many workers who have attempted to apply purification technics to rabies-infected brain tissues. The explanations for the contrasting behavior of rabies-infected brain and submaxillary gland suspensions with respect to Attasorb may reside in (1) function of the viscosity of the gland preparations and (2) the possibility that submaxillary gland may, in addition to the elementary rabies virus moiety, contain a particle of uniform size and weight (fig. 3) associated with infectivity, not previously recognized.

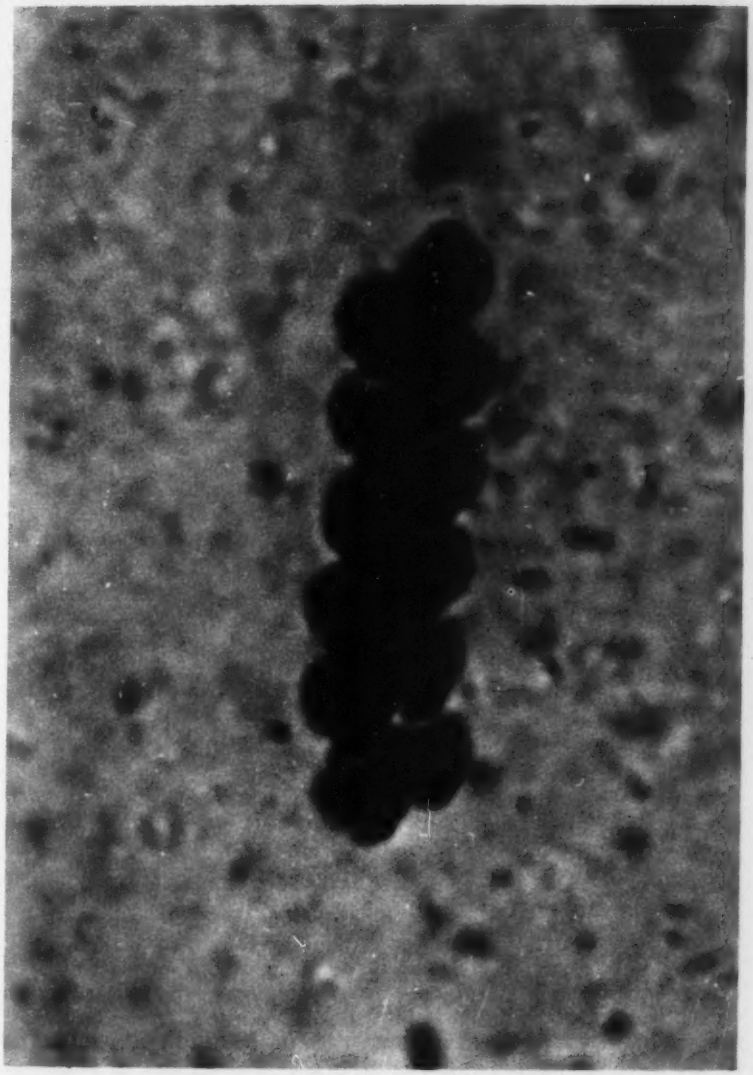


Figure 3. Agglomerated corpuscular elements from submaxillary salivary gland of a rabid dog. Microscopic bodies derive from sedimentable fraction by technic of incubation in 1:1 antirabies serum-phosphate buffer, followed by centrifugation for 1 hour at 5,000 r. p. m. at 5°C. Film of wet sediment is spread on slide, air-dried, and fixed with methanol. Staining is achieved by use of a mixture of pyronin B and methyl green basic dyes. Photographed with tungsten light source. (× 4860)

Investigations of rabid dogs by many workers have disclosed that possibly two important sites are parasitized by the rabies virus entity, namely, the central nervous system and salivary glands. The background of current fundamental knowledge of rabies virus and the disease which it induces in animals and man lies in basic studies of infected nerve cells. The cells of parasitized salivary glands in rabies are susceptible to disruption by mechanical means. Thus with aid of a simple mechanical device such as a Ten Broeck grinder, cell walls and nuclear membranes of parasitized submaxillary gland cells are disintegrated.

Consistent with modern concept of the structure of viruses, infected cells accumulate pathologic products in the form of virus proteins possessing property of antigenicity, and virus nucleic acids carrying the determinant of infection.[21] Nucleoproteins produced in cells during virus infection have been interpreted to represent the metabolizing and reproducing virus particles. According to Hyden, in the central nervous system the Purkinje cell's nucleolus is a principal site of attack of the rabies virus. The nucleolus is, in fact, a high-molecular weight entity, enormously enriched in ribonucleic acid. The nucleolus and hypothetical particles of the order of size greater than nucleolus but smaller than nucleus might conceivably be spared disintegration by a tissue grinder. If indeed Attaclay SF possesses remarkable sorptive property and capacity to reject nucleic acids,[20] it should in submaxillary gland suspension, assisted by the natural property of viscosity, repel high-molecular weight infective nucleotides, or previously undescribed virus-parasitized, nucleotide-containing particles of uniform magnitude and weight.

In support of the latter hypothesis it has been demonstrated that a microscopic, 2 to 2.5- by 2.5-micron size corpuscular element is present in post-Attasorb submaxillary gland suspensions of rabid dogs.[15] This particle, recoverable in the sedimentable fraction of the submaxillary gland, is shown to be associated with high infectiousness (table 2, and figs. 1 and 2).

The biological events relating to rabies virus' maturation and development during incubation in an injured host are not fully understood. Invasion of an infective particle or particles of rabies from an animal bite requires a prolonged and characteristic variable period of tissue migration. The infective moiety must reach a preferential site or sites within the host. Optimal nutritional environment is indispensable, if the determinant of infection is to mature and multiply sufficiently to attack the vulnerable central nervous system of the host. What is the exact route or routes of the viral particles' tissue migration? Is it, in naturally acquired disease, restricted to transport along nerve pathways as has been hypothesized?[22] This raises the question of an alternative route of migration of salivary gland particles of elementary or larger size, toward an environment particularly

suited for the virus' multiplication and development and survival in nature. The alternative preferential site in natural disease may be hypothesized to be the salivary glands, with the submaxillary gland, because of its richness in mucus- as well as serous-secreting cells, as an optimal site favoring virus self-reproduction and survival.

It has been the purpose of this article to point out that the site of the submaxillary salivary gland of naturally rabid dogs and other members of carnivoral reservoir hosts possesses potential for comprehensive fundamental and practical studies of rabies. Basic studies pertaining to relationship of the corpuscular elements of mandibular salivary glands of rabid dogs and rabies virus are now in progress at this laboratory.

SUMMARY

Concentration of determinant of infection of submaxillary glands of dogs with naturally acquired rabies disease has been accomplished. A chemically inert, natural-occurring, siliceous material, endowed with a sorptive characteristic was used in the separation of nonspecific nitrogen component from mechanically disrupted submaxillary glands. Subsequently, high-speed centrifugation of submaxillary gland suspension yielded a pellet of gelatinous substance. The latter material, termed sedimentable fraction, when derived from the submaxillary gland infected with rabies virus, retained ability to incite rabies disease experimentally; in addition, the fraction contained a corpuscular element reactive in antirabies serum not previously associated with naturally acquired rabies in dogs.

The background of present knowledge of the nature and properties of rabies virus has been discussed. Fundamental studies of nerve cells of animals infected with rabies have yielded much of known basic facts. Attention has been focused on the fact that mandibular salivary glands of rabid dogs may contain tenfold to a hundredfold more infective rabies LD_{50} for mice than an equivalent unit of fresh weight brain tissue. Yet, it is remarkable that corresponding fundamental knowledge of the relation of the host's salivary gland cells and rabies virus has been virtually neglected. This report raises the question of whether the site of the submaxillary salivary gland of naturally rabid dogs possesses potential for comprehensive, fundamental, and practical studies of rabies.

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THE FIFTH COLUMN AT THE BATTLE OF GETTYSBURG

RUDOLPH MARX, *M. D.*

UP to the beginning of the twentieth century losses from disease in most wars far outnumbered battle casualties. Various forms of painful infectious diarrhea, commonly called dysentery, have accompanied all armies as a fifth column, and have often decided the outcome of wars.

According to the Greek historian, Herodotus, the army of Xerxes I, King of the Persians in 480 B. C. was seized by plague and dysentery on its retreat from the battle of Salamis, and a great part of it destroyed. This catastrophe facilitated the crucial victory of the Greeks over the remainder of the Persian army left under the leadership of Mardonius in the following year at the battle of Plataea.

The failure of the Crusades was not brought about so much by the scimitars of the Saracens as by the toxins of hostile bacteria of dysentery and other epidemics. In 1227 A. D. dysentery turned back the army of Emperor Frederick II of Germany as he was crossing the Mediterranean on the way to Palestine. The same disease killed St. Louis of France in 1270 A. D., cutting short his second campaign to reach the Holy Land.

One of the decisive battles of history, the Battle of Valmy in 1792 between the French Revolutionary Army and the Royal Prussian Army, was won by dysentery when it attacked the Prussians, forced them to retreat, and saved the French Revolution at its birth.

Many similar examples in the medical history of wars could be added, and they reveal the peculiar phenomenon that dysentery usually plagued more the armies invading another country than it did the soldiers defending their homeland. Several explanations can be offered; one is that an invading army frequently has greater difficulties in maintaining a regular and adequate food supply. When an army has to live from the land, the supply of food is uncertain and often irregular. The invading soldiers fre-

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quently experience feast alternating with famine, and eat foods to which they are not accustomed. These factors can contribute to gastrointestinal upsets, weakening the natural defense against the overgrowth of harmful bacteria.

Another consideration is that just as different geographic areas have different flora and fauna, so each soil breeds different crops of bacteria and viruses. The food and water of every region has its own chemical peculiarities and contains different types of microorganisms. The longer a person lives in a certain locality, the more he becomes accustomed to the water of the region and becomes immunized to the specific mixture of the local microbes and viruses that are ever-present in the food, water, and air of his environment, and which even the most modern methods of sanitation cannot eradicate completely. The same minute forms of life which are usually innocuous to the resident are likely to be disease-producing in a newcomer entering from another bacterial milieu. These factors were more potent in former times before the advent of modern medicine and the knowledge of better purification and preservation of food and drink.

Diarrhea was so common in wars that it became the accepted belief that this condition was induced by fear of battle. In a number of realistic novels, scenes are described in which soldiers, when pinned down by enemy fire, soiled their pants. Medical experience has shown that in most men it was not the emotion of fear, but rather the sickness of dysentery, that produced these unfortunate accidents. The deathly paleness of the victims was more often caused by abdominal cramps and the weakening effects of dysentery than by fright. Modern army doctors have noted that under conditions of regular food supply and the absence of contagious dysentery, cases of diarrhea are the exception among soldiers during battle. Contrary to the popular notion, tension, anxiety, and fear will loosen the bowels only in a few men. In the majority, these emotional states will tend to "tighten them up" and cause constipation. The diarrheas of battles and of wars were usually produced not by fright but by bacteria.

During the American Civil War diarrhea and dysentery led all other diseases in the medical records of the United States Army. More than 25 per cent of all sickness reports were listed under this diagnosis. There were 1,739,135 recorded cases of acute and chronic diarrhea and dysentery, with 44,558 deaths among 2,780,000 Federal soldiers. As great as the occurrence rate of this disease was in the Federal Army, it was almost twice as high among the Confederate troops. According to available medical statistics compiled from comparative periods prior to July 1863, the annual number of cases of diarrhea and dysentery averaged 987 per 1,000 (many had repeated attacks) in the Confederate Army, compared with 543 per 1,000 in the Union Army. Such a high percentage of disablement weakened the effectiveness of the Confederate Army much more compared with that of

the Federal Army, since the Confederate Army was only half as numerous.

Most of the cases of dysentery occurred during the summer and fall months. There are indications that General Lee's troops suffered severely from dysenteric disorders in the early summer of 1863 during the Gettysburg campaign. The Confederate soldiers had been weakened by a bleak winter and a hungry spring spent in war ravaged northern Virginia. In April of that year, only 2 weeks before the battle of Chancellorsville, Lee complained to James A. Seddon, Secretary of War for the Confederacy, that the ration of his soldiers "consists of ¼ lb. of bacon, 18 oz. of flour, 10 lbs. of rice, to each 100 men about every third day, with some few peas, and a small amount of dried fruit occasionally as they can be obtained. This may give existence to the troops while idle but will certainly cause them to break down when called upon for exertion."

Such a diet was extremely short on calories and protein. Moreover, it was highly deficient in vitamins. Almost completely lacking was vitamin C, contained in fresh fruits and vegetables. This deficiency led to sporadic occurrences of scurvy among the army of northern Virginia, characterized in its classical form by spongy, bleeding gums, and internal hemorrhages. The more common, milder manifestations of vitamin C deficiency were mostly overlooked since they come on gradually and consist of vague symptoms such as fatigue, weakness, pain in the limbs, mental depression, and decreased resistance against infection.

Before the discovery of the vitamins a great part of the population of the temperate zone was suffering periodically from a certain degree of vitamin C deficiency. In the early spring of each year there was a critical period when the fruits and vegetables stored from the previous season were largely used up and the earliest edible green plants had not yet appeared. It has been assumed that the vague and euphemistic term, "spring fever" originated from the observation of this indisposition recurring each spring, a condition which in reality was caused by vitamin C deficiency.

At the time of the Civil War it was known empirically that fresh vegetables and fruits would cure the symptoms of scurvy. Therefore, at the first signs of spring, Confederate soldiers were sent out into the woods to collect sassafras buds, onions, and other wild vegetation.

The costly Confederate victory of Chancellorsville, 2 months before Gettysburg, was barren of strategic results and did not materially change the dismal picture of poor food supply of the army of Lee. According to Douglas S. Freeman, "of all the arguments that weighed with him" (Lee) in favor of the plan to invade Pennsylvania, "the most decisive single one was that he could

no longer feed his army on the Rappahannock. He had to invade the North for provisions regardless of all else." He had to seek food for his undernourished soldiers and fodder for his lean horses in the rich valleys of Pennsylvania, untouched by the war.

Their resistance lowered by months of malnutrition and lack of vitamins, the hungry soldiers of the South poured into the well stocked villages of Pennsylvania and gorged themselves with food and drink. They spoiled their stomachs, unaccustomed to such rich fare, and made their weakened gastrointestinal tracts readily susceptible to infection from the ever-present germs that cause dysentery.

An eyewitness account exists of a young officer named John Dooley who commanded the rear guard of Pickett's division on its approach to Gettysburg. He complains of his odious and difficult task of rounding up the numerous stragglers and sending them back to their companies. "For frequently it happens," he wrote, "that many soldiers leave their ranks through necessity, and weakened by diarrhea, can scarcely with all their efforts regain their ranks."

Freeman reports that fully 5,000 "sulkers and sick" contrived to march with the ambulance train from the battlefield of Gettysburg. This number would have represented approximately 10 per cent of Lee's army which was left to retreat. It is likely that a great number of these "sulkers and sick" were suffering from dysentery. A still greater percentage of the army probably suffered from milder forms of this highly contagious ailment, not sick enough to be excused from active duty during the emergency of a critical campaign, but weakened and discouraged by abdominal cramps and diarrhea, and their efficiency as officers and soldiers greatly impaired.

If the invasion of Pennsylvania was motivated by lack of food, the desperate need of shoes for the Confederate soldiers, often marching on gaping soles or on bare, bleeding feet, brought on the Battle of Gettysburg. Napoleon asserted that it needed as slight a provocation as a dog-fight to touch off a battle between two hostile armies poised for action. It is well known that the search for shoes caused a Confederate division to collide with Federal cavalry at Gettysburg—and the battle for the shoes was on—expanding into the decisive Battle of Gettysburg, a battle which had not been planned on this location by either command.

History books discuss the question of the high strategy of the generals and their mistakes, and praise the heroism of the soldiers on both sides. They fail to mention the unromantic factor of comparative medical statistics which gave the North the edge. The army of Meade had a far superior medical service, a better system of sanitation, and was in much better physical condition than Lee's army. Meade fought on interior lines, assuring a regular food supply consisting of meat, bread, and dehydrated vegetables.

During July 1863, the army of the Potomac was less affected by dysentery than the army of northern Virginia.

Except for its one defeat at Antietam, Lee's army had won all previous major battles in spite of inferior numbers. It had made up for this deficiency by its superior mobility, greater offensive spirit, and superior leadership. It was a different story at Gettysburg when for once the Confederate Army started the battle with the added advantage of numerical superiority which it maintained until the late afternoon of the second day.

On the afternoon of the first day two Confederate corps attacked and overpowered an outnumbered contingent of Federal troops defending the town of Gettysburg, and took the town. A. P. Hill, commander of the victorious third corps who participated in this victory, was described as sick and very pale on this day. This could explain his apathy and failure to reorganize his troops for immediate pursuit of the Federal columns that were fleeing in disorder toward the key position of Cemetery Hill.

On the second day of the battle it took two divisions of Longstreet's corps, only a few miles distant, the greater part of the day to get in position for attack, giving Meade time to bring up his reserves, reinforce, and extend his line. In spite of this delay Wright's brigade succeeded late in the afternoon, following a desperate struggle, to break through the Federal main position east of the Emmittsburg Road. The battle would have been won if the attack had been supported by the adjoining brigades and co-ordinated with simultaneous attacks of the other corps. There was no such co-ordination of effort on this occasion, nor all during the Battle of Gettysburg. Posey's brigade next to the successful break-through gave only half-hearted support. Mahoney's brigade in close proximity stood idly by. Meade was able to bring up his reserves, force back the victorious troops, and close the dangerous gap. In the twilight of the same day Early took important Federal positions on Cemetery Hill but was compelled to evacuate them for lack of support from Rode's division at his right.

It is an unanswered question how much the weakening and demoralizing effect of dysentery might have contributed to the strange apathy displayed by officers and soldiers of the Confederate Army at such crucial points of the battle, and to the unaccountable hesitancy and lack of resolution of some of the commanders which caused fatal delays and allowed the chance for victory to slip away.

It appeared to some observers, and later critics, that Lee himself did not show during the Battle of Gettysburg his usual energy, generalship, and good judgment which he exhibited in previous and in later battles. An eyewitness account of W. W. Blackford, officer on the staff of Jeb Stuart, claims that the commanding general was seriously handicapped in his conduct

of the battle by an attack of severe diarrhea. In his memoirs, *War Years with Jeb Stuart,* Colonel W. W. Blackford described his experience as follows:

In the supreme hour of battle the Commander in Chief is the soul of an army, that is, if he is worthy of the army he commands. This being so, anything which affects his physical condition at that time must have a powerful influence upon events. We all know the desperately weakening power of severe diarrhea, and this General Lee had, as I know. When Stuart arrived upon the left flank on the morning of the 2nd (July), there followed several cavalry combats, and during the evening I was sent to General Lee to report what had happened. I found General Lee at his headquarters near the town, but he was in his tent and I was told by one of his staff that I could not see him; so I gave my report to this officer, either Venable or Taylor, I think it was, who took it in to General Lee, and then I sat half an hour with them telling of our expedition. When coming on errands of this kind before, I had usually given my report to General Lee in person, but on this occasion I supposed he was too busy to see me. I was a little surprised to see him come out of his tent hurriedly and go to the rear several times while I was there, and he walked so much as if he was weak and in pain that I asked one of the gentlemen present what was the matter with him, and he told me General Lee was suffering a good deal from an attack of diarrhea. This was the evening of the 2nd day's fight and the day before the final contest. Now who in such a condition would not be affected in vigor of both mind and body, and will not this account for several things which were behind time, or not pushed forward as they should have been the 3rd of July?

Douglas S. Freeman in his introduction to Blackford's memoirs, which he calls an "honest narrative," attempts to refute the contention that General Lee was sick during the Battle of Gettysburg, and states that no officer of Lee's own staff ever reported any illness of the General during the Gettysburg campaign. He challenges the trustworthiness of Blackford's account of such a fateful incident. From the viewpoint of a medical historian it is hard to understand how an intelligent man like Colonel Blackford, a responsible officer on the staff of Jeb Stuart, could have invented the vivid details surrounding Lee's sickness, which Blackford claims to have witnessed under the striking circumstances of a great battle, circumstances that would have made unforgettable the images which the memory received.

The possible explanation of this contradiction could be that diarrhea was so common in the Confederate Army at Gettysburg that it was not thought worth mentioning unless the victim was put completely out of action. The close associates of General Lee may have tended to ignore the embarrassing predicament of their idolized chief, more so as they unconsciously resented so unheroic a situation, and they tried to forget it.

Clinicopathologic Conference

U. S. Air Force Hospital, Wright-Patterson Air Force Base, Ohio*

COUGH, FEVER, AND CHEST PAIN

Summary of Clinical History. A 46-year-old widow was admitted to this hospital on 6 March 1956 because of a cough productive of yellow sputum, fever, and chest pain of one month's duration. She died in this hospital 13 days after admission, on 19 March. The history obtained on admission revealed that the patient had been seen by her private physician approximately 3 weeks previously, and a roentgenogram of her chest on 20 February had been reported as normal. She had received one injection of penicillin approximately 2 weeks before admission and developed urticaria. One week prior to admission she had experienced chills, right-sided pleuritic pain, and increased cough. There was no history of hemoptysis or of recent weight loss.

There was a history of "allergy" of unstated type to penicillin and sulfonamides. She was a moderate smoker (20 cigarettes daily) and used alcohol occasionally. One episode of bronchopneumonia had occurred in 1950. She had toxemia of pregnancy 9 years prior to admission, and labor had been induced for that reason. Following this delivery the patient was said to have experienced "occasional renal difficulty" of an undescribed character.

Physical Examination. On admission the patient's pulse rate was 88 per minute; temperature, 103°F; and her blood pressure was 120/60 mm Hg. She was well developed and well nourished. She appeared to be acutely ill. No cyanosis or respiratory distress was noted. There was decreased excursion and diminution of breath sounds over the right side of the chest. No rales or pleural

*At the time of this conference, Col. Edward L. Olson, USAF (MC), was Commanding Officer. From the Laboratory Service Branch, Hospital Services Division, Maj. David Auld, Chief.

rubs were heard. The heart was normal in size and there were no murmurs. No abnormalities were noted on abdominal examination. Mild varicosities of the legs were present.

Laboratory Studies. Initial laboratory work showed the hemoglobin to be 14.0 grams per 100 ml; sedimentation rate, 30 mm per hour; white blood cell count, 5,250 per μl, with 68 per cent neutrophils and 31 per cent lymphocytes. Urinalysis showed a specific gravity of 1.020, 2 plus proteinuria, and no sugar. Microscopic examination of the urine showed occasional red and white blood cells. The blood urea nitrogen was 13 mg per 100 ml. A roentgenogram of the chest showed bilateral clouding of the lower lung fields and evidence of fluid in the right side of chest. Repeat blood studies on 9 March showed the white cell count to be 21,750 per μl, with 95 per cent neutrophils, 23 per cent of which were band forms.

Course in Hospital. The patient was placed on tetracycline initially, at an oral dosage of 0.5 gram every six hours. On 7 March bronchoscopy was attempted, but a great deal of purulent exudate was encountered in the entire tracheobronchial tree, and severe coughing developed. The air passages could not be visualized satisfactorily. A thoracotomy was performed on 7 March under local anesthesia to establish drainage of the right pleural space. At operation the right lower lobe appeared atelectatic. Culture of a small amount of fluid removed from the right pleural space yielded a growth of hemolytic *Staphylococcus aureus (Micrococcus pyogenes* var. *aureus).* The abundance of these organisms in the culture was not stated. The patient did poorly and the bilateral pulmonary infiltration extended, as shown by repeated roentgenographic examination of the chest. On 8 March, therapy was altered to include erythromycin and Chloromycetin (brand of chloramphenicol) by mouth, and bacitracin via nebulizer. Respiratory distress and laryngeal obstruction developed. A tracheotomy was performed on 10 March, and antibiotic therapy was altered to consist of 0.75 gram of streptomycin, 1 gram of Chloromycetin, and 200 mg of erythromycin, each every six hours intramuscularly; and 10,000 units of bacitracin, via nebulizer, every six hours.

On 14 March the urine output diminished to 300 ml and gross hematuria was noted. The maximum urine output from this time until death did not exceed 500 ml in any 24-hour period. On 14 March 500 ml of whole blood was administered, and streptomycin and bacitracin were stopped at that time. On 16 March many rales were audible in the chest and 2 plus pitting edema of the ankles was noted. The patient was then digitalized. On 17 March Chloromycetin therapy was stopped.

The patient continued to deteriorate slowly and an episode of cessation of pulse with no recordable blood pressure occurred at 1615 hours on 18 March. After this episode, the blood pressure fluctuated between 50/0 and 160/40 mm Hg, despite the intravenous use of norepinephrine, and the patient died at 1655 hours on 19 March.

Laboratory studies on the day of death revealed a hemoglobin of 10.2 grams per 100 ml, and a white blood cell count of 19,300 per μl, with 57 per cent band forms. Blood chemistry studies revealed the following values: sodium, 125 mEq per liter; chloride, 106 mEq per liter; blood urea nitrogen, 97.5 mg per 100 ml; calcium, 9.0 mg per 100 ml; and phosphorus, 11.4 mg per 100 ml. Carbon dioxide content was 38 volumes per cent. Urinalysis showed a specific gravity of 1.010 and 4 plus proteinuria, and innumerable red blood cells were noted in the microscopic examination.

DISCUSSION

Doctor Shirley:* To review the salient facts of the protocol, we have a 46-year-old woman who died after a 6 week illness. One month prior to admission, fever, chest pain, and a cough productive of yellow sputum began. She received one injection of penicillin about two weeks after the onset of her illness, which caused urticaria, so she received no more penicillin. Three weeks prior to admission, after one week of illness, the roentgenogram of her chest was interpreted as normal. A week before admission there was increased cough, chills, and pleuritic pain in the right side of the chest.

The initial laboratory work showed a markedly elevated sedimentation rate of 50. The white blood cell count, 5,250, which was normal, showed 60 per cent neutrophils and 31 per cent lymphocytes, indicating an infectious process with little total white cell response. There was no eosinophilia. The specific gravity of the urine was 1.020, which combined with 2 plus proteinuria shows that the kidneys were not functioning too well. Three days later she had 21,000 white blood cells per μl, with 75 per cent neutrophils; 23 per cent of these neutrophils were band forms. The blood urea nitrogen was normal. Admission chest roentgenogram showed clouding of the lower lung fields and some fluid in the right side of the chest. The day after admission, bronchoscopy was attempted but not completed because of a large volume of pus in the tracheobronchial tree, and the inducing of a severe cough in the patient. A thoracotomy was performed to drain the right pleural space. Only a small amount of fluid was collected. At that time the right lower lobe was atelectatic. Hemolytic *Staphylococcus aureus* was grown from the fluid obtained. The patient was treated with various antibiotics and benefited little from them. On 14 March, 8 days after admission, she began to go into renal failure. The urine output dropped to 300 ml and hematuria was noted. From then until she died, her urine output was not over 500 ml per day. On 16 March rales throughout the chest and 2 plus edema of the ankles were noted. The patient was digitalized. Two days later the pulse and blood pressure dropped suddenly and support with norepinephrine became necessary. In spite of this therapy, she died the following day.

Final laboratory studies indicated the carbon dioxide was decreased to 38 volume per cent; the blood phosphorus was elevated; and a uri-

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*Maj. Robert E. Shirley, USAF (MC), Resident in Aviation Medicine.

nalysis showed a 1.010 specific gravity, and 4 plus proteinuria with many red cells.

Past history on this patient is rather sparse—one episode of bronchopneumonia in 1950 and toxemia of pregnancy about 1947 or 1948, following which she had occasional renal difficulty, the nature of which is not known.

There are several things which can produce a similar course of disease as seen in this patient. If the first roentgenogram, taken by her private physician, was entirely normal, one must rule out a pulmonary neoplasm, with infection due to a staphylococcus arising peripheral to the neoplasm. A second diagnosis, which is more probable, is an acute *Staphylococcus aureus* pneumonia, probably present at the onset of illness. The course of staphylococcal pneumonia can be two types: First, a fulminating disease starting with an acute onset, temperature of about 101°F, tachycardia, marked leukocytosis, and toxemia, terminating in three to four days with signs of cerebral and gastrointestinal irritation. This is invariably fatal in a short time. The second, more usual, type of staphylococcal pneumonia which could occur results in metastatic abscesses in almost any part of the body, but usually in the lungs, kidneys, heart, or bones. Occasionally endocarditis is present, with skin rash or actual abscess formation. The symptoms which occur generally are related to the presence of an abscess in some organ giving symptoms related to the involved organ.

It is possible that this patient had "weak lungs" as a residual of bronchopneumonia in 1950. At the onset of her present illness she had a low-grade bronchopneumonia, chills, fever, and pain in the right side of her chest, but no roentgenographic changes were noted after a week. Because of her penicillin allergy, she may have had inadequate antibiotic therapy. The severe illness which began a week prior to admission would have then been caused by abscess formation in the right side of the chest which may have ruptured and produced a bacteremia, seeding infection in all portions of her body. Fulminating, rapidly fatal, irreversible changes occurred in the lungs, heart, kidney, and probably brain and bones.

Another type of staphylococcal infection that may occur is a metastatic infection from osteomyelitis or sinusitis. We have no indication of any such source here except for a notation of varicose veins in her legs. No mention was made that these were tender or painful upon movement of her extremities. This type of blood-borne staphylococcal infection would rapidly prostrate the patient and would resemble the last two weeks of the patient's illness. I cannot explain this patient's original white blood cell count of 5,250. The rest of her signs and symptoms follow a rather logical sequence of unresolved *Staphylococcus aureus* bronchopneumonia, followed by bacteremia, resulting in multiple abscesses, particularly in the kidney, possibly also in the heart. The end result was a terminal cardiac decompensation and uremia. The protocol gives no evidence of multiple abscesses in the brain.

Doctor Flanagan:* Would you care to make an exact diagnosis for the cause of renal failure? It is your impression that it was due to multiple renal abscesses?

Doctor Shirley: Yes.

Doctor Flanagan: Dr. Randolph, would you like to show the roentgenograms?

Doctor Randolph:** Serial films dating from the day of admission to the day of death are available on this patient. The admission film reveals consolidation of the right lower lobe, possibly also with some involvement of the right middle lobe. There is infiltration in the left lower lobe. Mediastinal structures are normal and the trachea is in the midline. This patient has an azygos lobe. A small amount of pleural fluid also is present on the right side. A thoracotomy was performed on the day following admission. This film shows a new finding on the left—an infiltration in the left mid-lung field, and by its distribution suggests it is in the superior division of the left lower lobe, which in turn suggests aspiration pneumonia. A second film, made on the same day, shows the upper portion of the abdomen. Although it doesn't completely exclude the diagnosis of subdiaphragmatic or liver abscess which has ruptured up through the diaphragm, it does not support it. On 9 March we see a finding which was not present before. In the upper portion of this consolidated right lower lobe, there is an ovoid translucency which could well be a small abscess, or, because the patient has had a thoracotomy, could possibly represent a loculated air bubble. On a film three days later, after the patient has had a tracheotomy, there has been increase in size of this translucency. Other changes have occurred: a clearing of the left mid-lung infiltration, the azygos vein has increased in size from that seen on the first film, and there are small patchy infiltrations in the right upper lobe which suggest pulmonary edema. The protocol tells us this patient began to go into cardiac failure, and this dilatation of the azygos vein is compatible with it. Later examinations show progression of the pulmonary edema. No clear-cut abscess is seen on the later films, nor on the film of the day of death.

The roentgenographic findings are compatible with the diagnosis of staphylococcal pneumonia. Other possibilities are massive septic pulmonary embolus, an aspiration pneumonia, or a subdiaphragmatic abscess extending into the chest.

A Physician: You say that a subdiaphragmatic abscess is still a possibility? Did you not say the film of the abdomen ruled it out?

Doctor Randolph: I saw nothing on the film of the abdomen to suggest a subdiaphragmatic abscess; let's put it that way.

Doctor Schalet:*** This patient presents a few very interesting problems. The most important is the question of primary cause of the disease.

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*Capt. John F. Flanagan, USAF (MC), Pathologist.
**Capt. Charles L. Randolph, Jr., USAF (MC), Radiologist.
***Capt. Norbert Schalet, USAF (MC), Medical Service.

Then, the cause and mechanism of her renal involvement, the relationship of that renal involvement to her previous history of toxemia of pregnancy, and the treatment she received in the hospital. This patient was a 46-year-old woman who apparently had no major illnesses prior to her admission to the hospital with an acute illness. Her illness lasted only 12 days. What diseases could cause such a rapid demise? The first and most important is pneumonia, as Dr. Shirley mentioned. There are two types of pneumonia that would present problems both in diagnosis and therapy. One notorious example is staphylococcal pneumonia. This pneumonia is seen in debilitated persons or as a secondary pneumonia in patients with malignant disease. I should mention that perhaps the explanation for the first normal white blood cell count that Dr. Shirley referred to, followed by an increased leukocytosis during the patient's course of illness, could be found in a secondary staphylococcal pneumonia, after influenza. A virus disease followed by a secondary infection would cause such a leukocytotic response.

Another type of severe pneumonia is due to Friedländer's bacillus (*Klebsiella pneumoniae*). This occurs more frequently in males than in females, especially in alcoholics. Frequently it is an aspiration pneumonia, but the description of the sputum given in the protocol does not fit the jellylike, tenacious, and purulent appearance of the sputum seen in Friedländer's pneumonia. I don't know whether a sputum smear was made during the patient's stay in the hospital, but usually there isn't much difficulty in demonstrating Friedländer's bacilli in the patient's sputum. Mycotic pneumonias also occur frequently in people who are debilitated by malignant diseases, but usually do not have a course as rapid with high fever, but rather a prolonged course. This woman did not have a debilitating disease in the past; therefore, I would rule out these possibilities. Vascular disease of the lung due to multiple emboli or massive infarction can be mentioned, but there are no peripheral signs or any inflammatory diseases of the lower extremities which might be the source of an embolus of the lung, nor a history of hemoptysis which usually accompanies lung embolization. In a massive pulmonary infarction the history would be much more dramatic as far as her respiratory distress is concerned. Another vascular disease—periarteritis—usually has a more prolonged course. It would not cause this type of picture and also would be accompanied by renal changes and by high blood pressure. Wagner's type of granulomatosis is accompanied by ulceration of the nose and sinusitis which this patient did not have. Malignancy also was mentioned before, and here alveolar cell carcinoma should be mentioned. The course usually is not so rapid as was the case in this woman. I don't know her reason for going to a physician six weeks before she entered the hospital. Interstitial fibrosis of Hamman and Rich[1] usually has a slow, progressive course, although a case of rapidly fatal Hamman and Rich type of interstitial fibrosis with necrosis has been reported.[2] That patient was much more cyanotic and her fever ran a course different from that seen in our patient.

Tuberculous pneumonia, even if it is fatal, is more slowly progressive, and there are only a few records showing leukocytosis as high as this woman had. As to her treatment in the hospital—I wonder whether it is wise to bronchoscope a patient who has such a high

fever and is in respiratory distress, unless there is a definite sign that removal of a foreign body is required, because it may add more to the already present respiratory distress. I believe the same is true for a thoracotomy, which is apt to induce a pneumothorax, again adding to the respiratory difficulties. A great part of such a patient's lung is then out of circulation and the blood cannot be oxygenated. Perhaps oxygen treatment under careful management, keeping in mind oxygen intoxication in anoxic patients, would be the therapy of choice. The variety of antibiotics that this woman received emphasizes the fact that we do not have adequate therapy for staphylococcus infections in general, especially those occurring in hospitals. *Staphylococcus aureus* was cultured from this patient's pleural fluid and it is a notoriously difficult bacteria to eliminate.

It is interesting to note that this woman had renal difficulty nine years prior to her admission. What type of renal difficulty did she have? Usually toxemias of pregnancy are not renal illnesses per se, although patients have convulsions and albuminuria. Teel and Reid[3] reported 80 such cases in which the patients recovered completely after their delivery. Usually they don't have any more difficulty, except when toxemia of pregnancy occurs in a patient with underlying hypertension or pyelonephritis or other renal disease. Therefore, I would not attach any significance to the past history of renal difficulties in this woman if her present illness hadn't terminated in renal failure. I don't think that she had renal disease on admission to the hospital.

Now, some comments about her death and treatment in her final days. There are two conditions that might cause the symptoms she presented during her last few days of life. In an overwhelming toxemia one must think of acute renal insufficiency, a type of acute tubular necrosis. Another condition that might have caused her renal symptoms is a low sodium syndrome causing a state of shock and a fall of renal output. Another possibility is bilateral adrenal insufficiency with hemorrhage, inasmuch as the laboratory data show she did have a low sodium level with hypotension. In summary, my final diagnosis would be staphylococcal pneumonia with bilateral adrenal hemorrhage. I don't know the cause of her heart failure; perhaps overhydration with low urinary output, general respiratory distress, and infection could produce heart failure in a woman who did not have previous heart disease.

Doctor Flanagan: Dr. Tobin, would you like to make a comment on this case?

Doctor Tobin:* First I would like to defend doing a bronchoscopy in a patient with staphylococcal pneumonia. These patients sometimes get a thick exudate they just can't cough up. I remember one case in which bronchoscopy extracted sort of a cast of the bronchial tree composed of thickened fibrous mucus, and the patient obtained relief even though he was very sick at the time. I thought it well worth doing. No one mentioned bacitracin as a possible contribution to this patient's renal damage. It was used, I'm sure, because the doctors were des-

*Col. James L. Tobin, USAF (MC), Chief, Professional Services.

perate to find something to combat the staphylococci. Using it as an inhalant gives rapid absorption, and once it is absorbed one is apt to get kidney damage. It was about three days after treatment with bacitracin that the urine output fell. I blame bacitracin for this. I think renal failure was the immediate cause of death. Staphylococcal pneumonias probably have a 75 per cent mortality rate. They can occur in young men who are not debilitated and result in very rapid death. The patients who recover often have permanent fibrotic damage of the lung.

Doctor Flanagan: Would you like to say something about renal failure or what the basis might be?

Doctor Tobin: I thought the basis was the toxic effect of bacitracin. The patient previously had some albuminuria which might have been explained on the basis of fever. I think the real difficulty started after she received bacitracin, and most of the damage was due to it.

Doctor McKay:* One thing no one seems to have considered is the possibility that this disease initially was primarily in the kidney and secondarily in the lung. I'll grant that the initial urinalysis was not spectacular but I don't think anyone would question that it was abnormal. Albumin and red and white blood cells were found, which could very well go along with a chronic kidney disease, probably a chronic, low-grade pyelonephritis which then erupted by hematogenous means and resulted in her death. The possibility of the kidney lesion being the initial lesion definitely should be considered.

Dr. Shirley's diagnosis:

Staphylococcal pneumonia with metastatic kidney abscesses

Dr. Schalet's diagnosis:

Staphylococcal pneumonia with adrenal failure and acute renal failure secondary to acute tubular necrosis

PATHOLOGIC FINDINGS

Doctor Flanagan: At autopsy the body was that of a moderately obese 46-year-old woman. A few ecchymotic areas were present over the antecubital fossae, and a slight degree of pitting edema was seen over the tibia, bilaterally. External examination also showed the poorly healed right thoracotomy incision which broke open by gentle manipulation. There was a tracheotomy incision in the neck. The lungs showed rather impressive findings: The entire tracheobronchial tree was almost completely filled with purulent debris and mucoid material, and the right lower and right middle lobes and the left lower lobe were bulky, firm, noncrepitant, and showed multiple areas of abscess formation surrounding the bronchi. These areas shelled out readily to reveal cavities which measured up to 2 cm in diameter. At this point I might say that the culture from one of these abscessed areas yielded a few colonies of hemolytic *Staphylococcus aureus*

*Maj. William M. McKay, Jr. USAF (MC), Anesthesiologist.

and a moderate growth of *Escherichia coli*. The upper lobes of the lungs showed a considerable degree of compensatory emphysema. There was a mild amount of pleural effusion on both sides of the chest, with a few adhesions of the pleura to the chest wall.

The adrenals were considerably enlarged and contained several large, discrete, golden nodules. There was a marked increase in weight of the adrenals, to 30 grams (normal weight, about 12 grams). The kidneys showed rather unexpected findings. They were enlarged to almost twice the normal weight (left, 316 grams; right, 270 grams). Both were swollen, quite pale, and showed multiple punctate subcapsular hemorrhages. The brain showed the presence of a cyst in the third ventricle, which will be described later.

I think first it would be well to discuss the fundamental infectious process in the lungs. Histologically the lungs presented a typical pattern of septic pneumonia with findings that indicate the process was of considerable duration. The right middle, right lower, and left lower lobes showed multiple large areas of abscess (fig. 1) formation

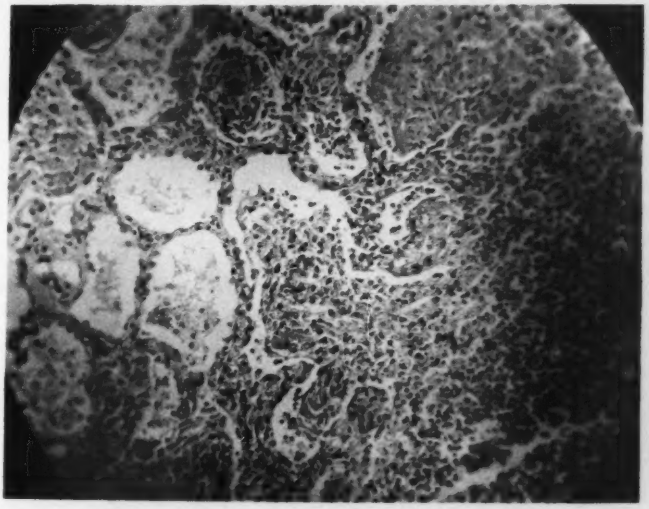

Figure 1. Section of lung showing edge of abscess.

distributed about the bronchi. In many cases these areas were enclosed in fairly well-developed, connective tissue capsules. Most of the small and medium-sized bronchi were filled with partially organized purulent material. Areas of pulmonary parenchyma not directly involved by abscess formation showed diffuse bronchopneumonia with extensive organization. No microorganisms could be identified in any of the sections.

This case serves to emphasize that *Staphylococcus aureus* is still an important cause of disease in men despite the widespread use of antibiotics. Rogers[4] collected highly significant data from experience,

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at the New York Hospital, with staphylococcal disease by examining the records for selected years in the pre- and post-antibiotic periods. During the period immediately before the development of penicillin, he found that the mortality rate in adults from staphylococcal bacteremia at the New York Hospital was 69 per cent, and in the period from 1949 to 1955 it was 70 per cent. More striking figures were seen in staphylococcal pneumonia, in which case the preantibiotic mortality was 30 per cent and the postantibiotic mortality rate was 92.3 per cent. Spink[5] stated that the mortality with staphylococcal septicemia remained (in 1954) at 50 to 90 per cent at the University of Minnesota, despite the use of antibiotics. In evaluating the importance of these studies one must consider the very significant fact that primary staphylococcal bacteremia and pneumonia were frequent in the preantibiotic period, while primary infection became much more rare in the antibiotic period, and the more common situation was to find staphylococcal infection as the terminal process in leukemia, carcinomatosis, uremia, and debilitating diseases of the aged. Despite this weighting of the mortality figure by the presence of serious primary disease, it is evident that staphylococcal infection is still a serious problem in patient management.

One of the prime factors in continued high mortality rates from staphylococcal disease is the emergence of antibiotic-resistant strains of the organism. In the early 1940's practically no strains of staphylococci were resistant in vitro to therapeutic doses of penicillin, while several studies[6] in the last few years have shown that at least 50 to 70 per cent of the staphylococci isolated in hospitals are resistant to penicillin. Erythromycin also has become less effective with the emergence of resistant strains. Antibiotic resistance in such a high percentage of the organisms makes it almost mandatory that one isolate the organism and determine antibiotic sensitivity before undertaking a long course of therapy.

What properties does the organism possess that make it such a serious invader of the body? Many questions are still unanswered; we know, however, that the organism in culture is capable of elaborating a complex group of toxins and metabolites which possibly assist the organism in setting up infection. Among these products are hemolysin, leukocidin, dermonecrotic toxin, fibrinolysin, hyaluronidase, and enterotoxin. All of these substances are more commonly found in pathogenic organisms isolated from infections in man than in the nonpathogenic strains, but the occurrence of these cellular products is highly variable and they may appear singly or altogether in a single strain. Within the past few years, the occurrence of coagulase in a strain has been accepted as one of the most reliable criteria of pathogenicity. Ninety to 95 per cent of pathogenic strains possess the ability to coagulate citrated rabbit or human plasma in 1 to 3 hours after inoculation of the plasma with an equal volume of broth culture. Teleologically one could reason that this ability to coagulate plasma serves to wall off the organism in the body and prevent contact between the organisms and the body defense mechanism. There is considerable evidence that the staphylococcus is able to achieve inaccessible foci in the tissues shortly after infection

so that the body defense mechanisms and antimicrobial agents act at a considerable disadvantage. Coagulase may play an important role in this sequestration of the organisms but the relationship is not yet clear. Coagulase and the toxic products of the staphylococci have largely been identified by their biologic effects, while their chemical structure and relative importance in determining the course of the disease are still unknown. So we see that even the major determinants of staphylococcal infection are poorly understood, and, what is more important, we see that deep staphylococcal infection even with the most enlightened therapy is still accompanied by a high mortality rate.

Histologic examination of the kidneys in our patient revealed an unusual complication of staphylococcal infection. The characteristic lesion in the kidneys consisted of anemia of the glomeruli, marked exudation of neutrophils in the glomeruli, hemorrhage into Bowman's space (fig. 2), marked dispersion and vacuolation of the cytoplasm

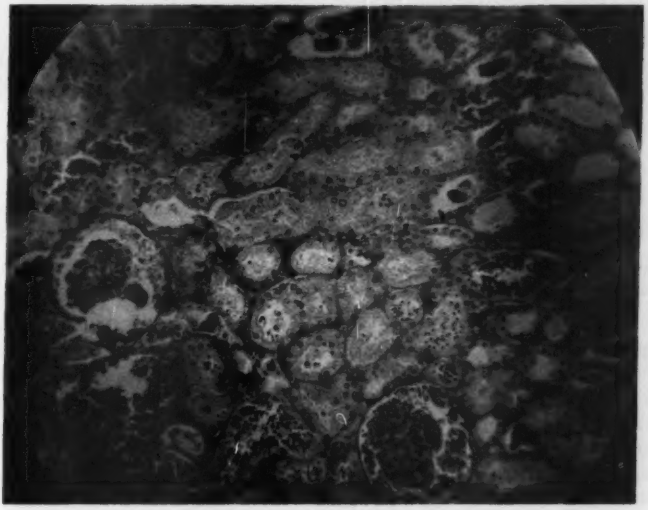

Figure 2. Section of the kidney showing hemorrhage into Bowman's space.

in the tubular cells, and many red blood cell casts in the tubules (fig. 3). Nearly every nephron showed at least some of these alterations. Careful examination showed no evidence of acute tubular necrosis or of toxic tubular damage. The most common organism associated with the development of glomerulonephritis is *Streptococcus pyogenes* of type 12 or type 4, with, at most, 1 or 2 per cent of acute glomerulonephritis being ascribed to staphylococcal infection. Nothing in the clinical history or hospital course of this patient indicated recent streptococcal infection and it seems reasonable to assume that the renal disease was an outcome of staphylococcal infection in this case.

Histologically the adrenal glands showed marked thickening of the cortical layer by diffuse hyperplasia and by the presence of multiple unencapsulated nodules composed of cortical cells. We are unable to arrive at any conclusion regarding the level of hormone production by the hyperplastic adrenals inasmuch as no satisfactory histochemical method of tissue adrenal hormone assay is available.

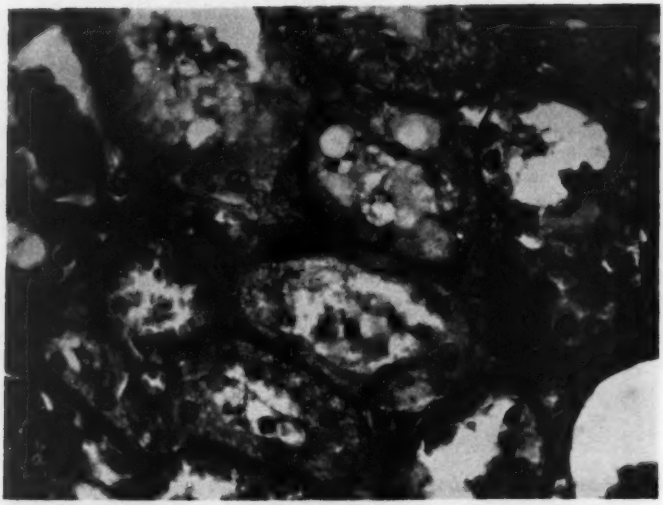

Figure 3. Section of kidney showing marked dispersion and vacuolation of the cytoplasm in the tubular cells, and blood cell casts in the tubules.

Examination of the brain showed a 1.5-cm cyst in the third ventricle which was lined by a thin layer of cuboidal epithelium and was filled with colloid material. Colloid cysts of this character arise from a persistent fetal structure, the paraphysis, which is more highly developed in the third ventricle of lower animals. These cysts may occasionally function as ball valves obstructing the outflow of cerebrospinal fluid through the aqueduct of Sylvius or the interventricular foramina and producing paroxysmal headaches. The headaches may sometime be relieved by changing the position of the head. The cyst in this particular case could never have functioned as a valve because it was firmly attached at several points to the wall of the third ventricle and was not suspended by a single slender stalk as is the usual case.

In summary, we have a case of organizing bronchopneumonia accompanied by abscess formation involving extensive areas in both lungs. This infection was proved by ante-mortem and post-mortem cultures to be due to hemolytic *Staphylococcus aureus*. The pulmonary infection was complicated by the development of acute diffuse glomerulonephritis. Nodular hyperplasia of the adrenals and colloid cyst of the third ventricle were incidental findings.

Pathologic Diagnoses:

1. Bronchopneumonia, organizing, with abscess formation due to *Staphylococcus aureus (Micrococcus pyogenes* var. *aureus)*
2. Acute diffuse hemorrhagic glomerulonephritis
3. Nodular hyperplasia of the adrenals
4. Colloid cyst of the third ventricle

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PLIGHT OF THE CAPTURED AIRMAN

Special survival training, including training that prepares a man for the eventuality of becoming a prisoner of war, is a by-product of modern warfare and is a particular problem of the Air Force. Flying over enemy territory with the constant hazard of forced landing or parachute escape from a damaged aircraft, the airman continually faces a potential survival situation. Yet ordinary basic training gives him only rudimentary skills in the use of weapons for self-defense. It does not condition him for long marches and highly uncomfortable bivouacs. The flyer has not had any close contact with the enemy or his terrain; he is not toughened by the rigors of ground warfare. There is a violent contrast between his combat situation in an aircraft and his survival situation on the ground. Furthermore, a future conflict may confront our airmen with the possibility of capture by an enemy who has developed psychological warfare to the greatest extent in history, and who continues to wage this warfare unrelentingly against the prisoner who falls into his hands.—Louis Jolyon West: Psychiatric Aspects of Training for Honorable Survival as a Prisoner of War. *The American Journal of Psychiatry,* October 1958.

GEN. TWITCHELL HEADS MILITARY SURGEONS, MEETING DEVOTED TO SPACE AGE PROBLEMS

Brigadier General Harold H. Twitchell, USAF (MC), surgeon of the U. S. Air Forces in Europe was installed as president of the Association of Military Surgeons at the close of the society's 65th annual meeting in Washington last month. He succeeded Colonel Charles R. Mueller, MC, USA (Ret.) of the Veterans Administration who presided at the convention which drew 2,000 delegates to hear papers devoted to the theme of "Dynamic Medicine and Rehabilitation in the Space Age."

General Twitchell

Lieutenant Colonel Carl W. Hughes, MC, USA, chief of general surgery at Tripler U. S. Army Hospital, won the Sir Henry Wellcome Medal and Prize for his essay on the subject of vascular surgery in the medical services. The Gorgas Medal, established by the Wyeth Laboratories for distinguished work ir preventive medicine, was awarded to Lieutenant Commander John H. Ebersole, MC, USN, medical officer of the U. S. S. *Seawolf,* who organized the medical departments of the first two nuclear-powered submarines.

The Major Louis Livingston Seaman Prize was given Colonel Rollin L. Bauchspies, MC, USA, commanding officer of the U. S. Army Hospital, Neubrucke, Germany, for his series of articles "The Courageous Medics of Anzio" that appeared in *Military Medicine.* The 1958 Stitt Award, established by the Pfizer Laboratories Division, Charles Pfizer and Company, in honor of the late Rear Admiral Edward Rhodes Stitt, was presented to Captain George L. Calvy, MC, USN, chief of medicine, U. S. Naval Hospital, St. Albans, N. Y. for his clinical research in the antibiotics combating staphylococcic pneumonia.

Colonel William H. Lawton, USAF (MC) (Ret.), who died on 6 November, had been chosen as the 1958 winner of the McLester Award

Col. Hughes

Cdr. Ebersole

Col. Bauchspies

from J. B. Roerig Company Division, Charles Pfizer and Company. The late Col. Lawton was honored for his work in hypoglycemia and its effect on the accident rate in the Air Force Training Command. The Founder's Medal was presented to Colonel George B. Green, USAF (MC), for his contributions to military medicine and for meritorious service to the Association.

The meeting opened with an address by General Alfred M. Gruenther, USA (Ret.), president of the American Red Cross, who was introduced by Major General Howard McC. Snyder, USA (Ret.), physician to the President. Major General George F. Lull, MC, USA (Ret.), assistant to the president of the American Medical Association, served as moderator of the panel of speakers which included the surgeons general of the military departments and the Public Health Service, and the chief medical director of the Veterans Administration. "The Changing Medicare Program" was the subject of a timely presentation by Brigadier General Floyd L. Wergeland, MC, USA, director of Medicare. Major General Wilford F. Hall, USAF (MC), served as chairman of a panel of experts for the discussion of "Occupational Health Problems in Space Flight," and Brigadier General Don Flickinger, USAF (MC), headed a symposium on "Problems of Space."

Capt. Calvy

Col. Lawton

Col. Green

ADM. RALPH MALONE RETIRES, SUCCEEDED AS NAVY DENTAL CHIEF BY ADM. SCHANTZ

Rear Admiral Ralph W. Malone, Assistant Chief of the Bureau of Medicine and Surgery for Dentistry and Chief of the Dental Division since March 1955, retired on 1 November after 32 years of service. He was succeeded by Rear Admiral Curtiss W. Schantz who has been commanding officer of the U. S. Naval Dental School, National Naval Medical Center, Bethesda, Maryland.

Admiral Malone

During his long service, Admiral Malone provided outstanding leadership and guidance to the Navy Dental Corps. He directed "Operation Build-up" that raised the strength of the regular corps to more than 1,000 officers and sponsored a training program in mass casualty care. In World War II, while assigned to the USS *New Jersey*, he participated in the campaigns at Kwajalein and Truk Islands. Earlier, on one of his three tours of duty as an instructor in crown and bridge dentistry at the Naval Dental School, he served as personal dentist for President Franklin D. Roosevelt.

Admiral Schantz

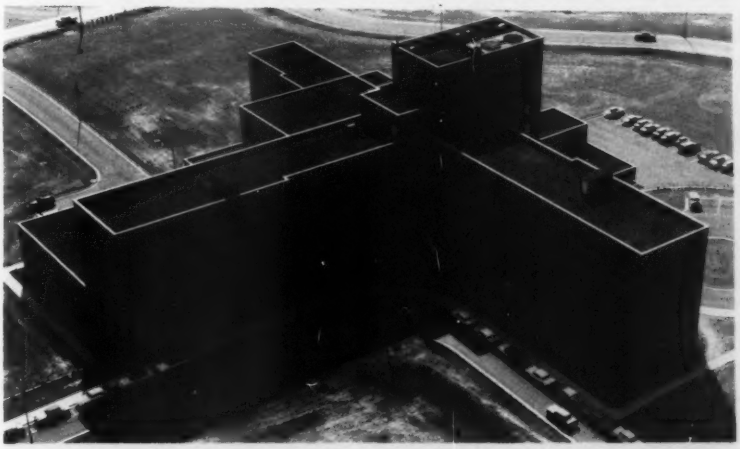
Admiral Schantz, in his 28 years of active duty, has had broad experience as a dentist, a leader, and as a dental educator. In World War II he headed the Dental Department of the Naval Training Center, Great Lakes, Illinois, where he organized and administered the largest group dental practice in existence with over 500 dental officers on duty. During the Korean Conflict, he served as fleet and force dental officer for the Pacific Fleet and supervised the rapid expansion of dental facilities to support the Marines in Korea, and the Fleet in the Far East.

On 31 October in an impressive change of command ceremony, Admiral Schantz, who was promoted on 1 November, relinquished command of the Naval Dental School to Captain Eric G. F. Pollard, DC, USN, his executive officer for the past two years. Guests attending the ceremony included Dr. Frank B. Berry, Assistant Secretary of Defense (Health and Medical), Major Generals James M. Epperly and James S. Cathroe, Army and Air Force dental chiefs, Dr. Clemens V. Rault, Dean, Georgetown University School of Dentistry, Dr. Bruno Floria, President, District of Columbia Dental Society, and Dr. C. Willard Camalier, American Dental Association.

KOSSUTH HEADS CIVILIAN MEDICAL GROUP

Colonel Kossuth

Colonel Louis C. Kossuth, USAF (MC), was installed recently as president of the American College of Preventive Medicine at its annual meeting in St. Louis. The Deputy Command Surgeon of the Air Defense Command is the first military officer on active duty to be chosen for this office. Members of the American College of Preventive Medicine, which was formed in 1954, represent the fields of public health, aviation medicine, and occupational medicine. Most military members are specialists in aviation medicine as is Dr. Kossuth, a flight surgeon, who has served in the Air Force since 1941. Prior to his present assignment, he was deputy commandant of the School of Aviation Medicine. He is a former chief of preventive medicine at USAF and USAFE Headquarters.

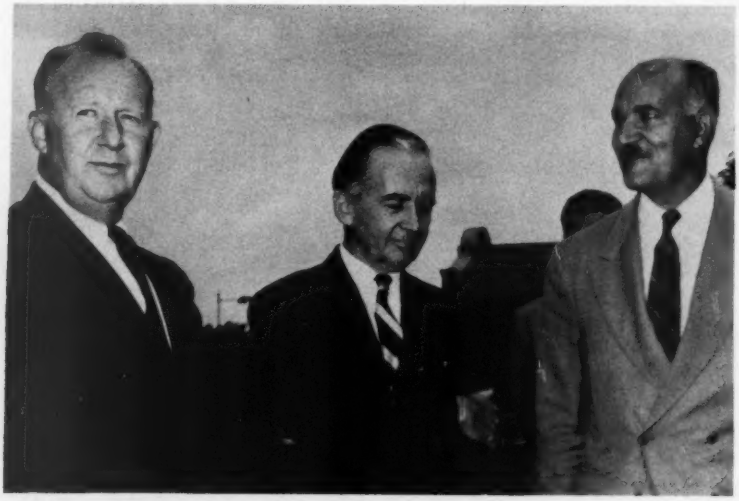


NEW ANDREWS AFB HOSPITAL. Here is the new 250-bed hospital at Andrews Air Force Base, Washington, D. C., which was dedicated in ceremonies on 7 November by Major General Olin F. McIlnay, deputy surgeon of the Air Force. The five-story air conditioned brick structure, which replaces the World War II hospital at Bolling Air Force Base, contains the most modern innovations for providing specialized medical care. Colonel Aubrey L. Jennings, USAF (MC) is the hospital commander.

ARMY NAMES NEW SPECIALIST CORPS CHIEF

Colonel Ruth A. Robinson has been named the fourth chief of the Army Medical Specialist Corps succeeding Lieutenant Colonel Harriet S. Lee, now assigned to the Army Medical Service Historical Unit at Walter Reed Army Medical Center. For the past three years, Colonel Robinson has been chief occupational therapist at Walter Reed Army Hospital and had previously served in the same capacity at Fitzsimons Army Hospital and Brooke Army Hospital. Before her appointment in 1948 to the former Women's Medical Specialist Corps, she had served in the Medical Department of the Army as a civilian. She is the immediate past president of the American Occupational Therapy Association.

Colonel Robinson



CAIRO CEREMONY. Captain John R. Seal, MC, USN (left), commanding officer of the Naval Medical Research Unit No. 3, Cairo, Egypt, was host at an "open house" recently to the Honorable Raymond A. Hare, United States Ambassador to the United Arab Republic, and Dr. Nur el Din Tarraf (right), chairman of the Executive Council, Egyptian Region, UAR, on the occasion of the tenth anniversary of the founding of the laboratory. More than 400 guests attended the ceremony.

GENERAL OGLE DECORATED. The retiring Surgeon General of the Air Force, Major General Dan C. Ogle (right), is shown receiving the Distinguished Service Medal in a Pentagon ceremony recently from General Thomas D. White, USAF, chief of staff of the Air Force, for "exceptionally meritorious service to the United States in a position of great responsibility." General Ogle, who retired on 30 November, has been succeeded by Major General Oliver K. Niess.

USAF COMMAND DENTAL SURGEONS MEET

Major General Olin F. McIlnay, Deputy Air Force Surgeon General, gave the welcoming address at the annual conference of the Air Force command dental surgeons in Washington, in mid-October. The conference was conducted by Brigadier General James S. Cathroe, Assistant for Dental Services, to discuss the policies and programs affecting dental service operations at command and base levels. Procedures for increasing the effectiveness of current educational, preventive dentistry, and research programs were discussed. Colonel Donald C. Hudson, chief of the Research Dentistry Division, Randolph Air Force Base, Texas, presented the results of investigations of ultra high-speed handpieces and auxiliary equipment for cavity preparation and these were recommended for all Air Force dental clinics.

Official Decorations

The following awards were recently announced by the Departments of the Army and the Air Force.

Distinguished Service Medal

Dan C. Ogle, Maj. Gen., USAF (MC)

Army Commendation Ribbon

Gilbert M. Eisner, Capt., USA, MC*

Charles L. Gilbert, Col., USA (MSC)

Air Force Commendation Medal

John F. Pierce, Lt. Col., USAFE (MSC)

Frederick S. Spiegel, Lt. Col., USAF (MSC)

John M. Rogers, Lt. Col., USAF (MSC)

*With metal pendant

DEATHS

SMITH, Stuart Gross, Major General, MC, USA, of Springfield, Ill.; Commanding General of Letterman Army Hospital, San Francisco, Calif.; graduated in 1927 from Washington University School of Medicine, St. Louis, Mo., served in an enlisted status from 18 October 1918 until 26 December 1918; commissioned a first lieutenant in the United States Army Medical Corps Reserve 3 March 1926; appointed a first lieutenant in the United States Army 15 March 1926 and ordered to active duty 6 April 1926; died 19 November 1958, age 58, at Fitzsimons Army Hospital, Denver, of cancer.

TEBOW, Louis Elliott, Captain, MC, USN, of Tamaroa, Ill.; assigned to U. S. Naval Hospital, Memphis, Tenn.; graduated in 1941 from the University of Illinois College of Medicine, Chicago, Ill.; appointed a lieutenant, junior grade, in the United States Navy 23 June 1941; died 27 October 1958, age 48, at the U. S. Naval Hospital, Memphis, of myocardial infarction.

TIPPS, Bill, Captain, USAF (MC), of Oklahoma City, Okla.; assigned to 7505th U. S. Air Force Hospital at Burderop Park, Eng.; graduated in 1956 from the University of Oklahoma School of Medicine, Oklahoma City, Okla.; commissioned a second lieutenant in the United States Air Force Reserve on 25 May 1952; ordered to active duty 26 June 1956; died 15 October 1958, age 28, at Princess Mary Hospital, RAF, Halton Aylesbury, England, of doriden poisoning.

CORRECTION

The new address for Brigadier General Thomas W. Mattingly, MC, USA, (Ret.) was incorrectly given in the September *Journal*. Dr. Mattingly is the Director of Medical Education of the Washington Hospital Center, 110 Irving Street, N. W., Washington, D. C.

A MESSAGE FROM THE A. M. A.

In October 1958 the Committee on Military Medical Affairs, one of the two Committees of the American Medical Association's Council on National Defense, met with the Assistant Secretary of Defense (Health and Medical) and his staff and the Director of the Selective Service System in regard to matters of mutual interest to the military services and American medicine.

One of the topics discussed was the expiration on 30 June 1959 of the Universal Military Training and Service Act of 1948, as amended. While every registrant who has been deferred has a general liability until he is 35 years old, irrespective of the expiration of the Act, the authority to induct registrants who have not been deferred would terminate if the Act is not extended.

Under the present amendment to the basic Act, which became effective 1 July 1957, the Department of Defense has been able to fill its requirements for medical officers from volunteers. The last draft call for physicians was in February 1957. Since that time, however, there has been a steady decline in the number of interns volunteering for deferment and subsequent active duty in the Armed Forces Reserve Medical Officer Commissioning and Residency Consideration Program, popularly known as the Berry Plan. This decline may be due to the fact that the young medical school graduate is aware that there is only one chance in two of having to serve, so he takes a chance on not being called.

Unless renewed interest in the Berry Plan is shown by interns, it now appears that a draft call for physicians will be necessary in fiscal year 1960 if the military services are to fulfill requirements for qualified medical officers.

Any extension of the general draft law, including the special provisions for the call up of physicians, will be considered by the 86th Congress which convenes next month. It appears that the Department of Defense and the Selective Service System will propose that the draft law be extended beyond its expiration date of 30 June 1959 in substantially its present form. It is believed that the obligation to serve, which the law provides, is necessary if the needs of the military services for medical officers are to be met by volunteers or otherwise.

With respect to the extension of the special provisions of the draft Act applicable to medical, dental, or allied specialists, the American Medical Association has recommended that the Act not be extended beyond its expiration date. The Association

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From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense. —*Editor*

believes, however, that if the demands for the defense and security of our nation are such that, in the opinion of the Congress, it is necessary to have a draft act to maintain the strength of our Armed Forces, then the extension of the present legislation would be one method to ensure sufficient physicians for the military services. This is a reaffirmation of the policy position of the Association which was adopted at the time the present amendment to the basic Act was proposed.

The Association also has recommended that there be retained the provisions of the law relating to the National Advisory Committee and the local and state committees which advise the Selective Service System in regard to the selection of needed medical, dental, and allied specialists required for service. These committees consider, among other factors, the needs of the civilian population in the communities from which the physicians would be taken.

In regard to specialist categories, moreover, the Association recommends, as it has on a previous occasion, that the word "other" should be substituted for the word "allied" wherever it appears in the law. This would authorize a selective call for all other specialists, such as engineers, mathematicians, physicists, et cetera, needed by the military forces. Presently, the law provides for the selective call up of medical, dental, or "allied" specialists.

The Association believes that physicians should be registered and classified in the same manner as other citizens in the same age group, deferred for educational purposes in the same manner as other registrants, and called to active duty or inducted under the same general provisions as other registrants deferred for educational purposes.

Major Anthony Borski Wins Kimbrough Award

Major Anthony A. Borski, MC, USA, Fitzsimons Army Hospital, Denver, Colorado, received the James C. Kimbrough Memorial Award for his presentations on "Evaluation of Renal Function Using Radioiodine Diodrast" and "Genitourinary Tuberculosis" at the annual urological seminar in honor of the late distinguished Army urologist, held at Brooke Army Hospital, San Antonio, Texas, 3-5 November. The award, consisting of an honorarium for new urological publications, is the gift of Mrs. Kimbrough. The extensive program of scientific papers and case reports was arranged by Colonel Louis K. Mantell, MC, USA, Chief of the Urological Service, at the hospital.

OFFICERS CERTIFIED BY SPECIALTY BOARDS

The Surgeons General of the military departments have announced certification by specialty boards of the following officers of the regular establishment since publication of listings in previous issues of the *Journal*.

American Board of Psychiatry and Neurology

Neurology

Henry W. Hogan, Maj., USA

American Board of Orthopedic Surgery

Paul W. Brown, Maj., USA

American Board of Urology

William A. Collins, Jr., Lt. Col., USA

Samuel Rodriguez, Lt. Col., USA

American Board of Internal Medicine

Gastroenterology

Norman M. Scott, Jr., Lt. Col., USA

American Board of Pathology

Pathologic Anatomy

Proctor L. Child, Maj., USA
Robert S. Cox, Jr., Capt., USA
James L. Hansen, Lt. Col., USA
James C. Hayes, Maj., USA
Paul W. Palmer, Maj., USA

Clinical Pathology

Robert S. Cox, Jr., Capt., USA

American Board of Otolaryngology

Harry W. McCurdy, Maj., USA

American Board of Surgery

Hu A. Blake, Lt. Col., USA
Oliver R. Buesing, Lt. Col., USA
Robert D. Pillsbury, Lt. Col., USA
Donald W. Robinson, Cdr., USN
Jack T. Rush, Lt. Col., USA

American Board of Oral Surgery

Leo Korchin, Lt. Col., USA

American Board of Prosthodontics

John F. Bowman, Capt., USN
Frank J. Kratochvil, Cdr., USN
Christopher E. Thomlinson, Jr., Capt., USN

American Board of Veterinary Public Health

William A. Bridenstine, Maj., USA

Harvey L. Rubin, Lt. Col., USA

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